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Illinois Natural History Survey, Urbana

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THE DEGRADATION OF SELECTED PESTICIDES IN SOIL:

A REVIEW OF THE PUBLISHED LITERATURE

MUNICIPAL ENVIRONMENTAL RESEARCH LABORATORY
OFFICE OF RESEARCH AND DEVELOPMENT
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This report contains a literature summary on the degradation of forty-five pesticides in soil. The point of beginning of each literature review is the year of issue of the patent for the particular pesticide. After compilation of the literature data for each pesticide, conclusions were formulated regarding the suitability of soil disposal of these pesticides. On the basis of the data collected in this report it was suggested that ten pesticides are suitable for soil disposal, twenty-one are not suitable for disposal, and the data for fourteen are insufficient to formulate any conclusions regarding their suitability for soil disposal.

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FOREWORD

The Environmental Protection Agency was created because of increasing public and government concern about the dangers of pollution to the health and welfare of the American people. Noxious air, foul water, and spoiled land are tragic testimony to the deterioration of our natural environment. The complexity of that environment and the interplay between its components require a concentrated and integrated attack on the problem.

Research and development is that necessary first step in problem solution and it involves defining the problem, measuring its impact, and searching for solutions. The Municipal Environmental Research Laboratory develops new and improved technology and systems for the prevention, treatment, and management of wastewater and solid and hazardous waste pollutant discharges from municipal and community sources, for the preservation and treatment of public drinking water supplies, and to minimize the adverse economic, social, health, and aesthetic effects of pollution. This publication is one of the products of that research; a most vital communications link between the research and the user community.

The following report contains a literature summary on the degradation of forty-five pesticides in soil. Some preliminary conclusions are presented as to the suitability of certain pesticides for soil disposal.

Francis T. Mayo, Director
Municipal Environmental Research
Laboratory

ABSTRACT

This report contains a literature summary on the degradation of forty-five pesticides in soil. The point of beginning of each literature review is the year of issue of the patent for the particular pesticide. After compilation of the literature data for each pesticide, conclusions were formulated regarding the suitability of soil disposal of these pesticides. On the basis of the data collected in this report it was suggested that ten pesticides are suitable for soil disposal, twenty-one are not suitable for disposal, and the data for fourteen are insufficient to formulate any conclusions regarding their suitability for soil disposal.

This report was submitted in fulfillment of Grant No. R 803591-01 by the Illinois Natural History Survey, University of Illinois, under the partial sponsorship of the U.S. Environmental Protection Agency. This report covers the period from February 1975 to May 1976.

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SECTION I INTRODUCTION

The use of pesticides has steadily increased in the thirty years since the introduction of DDT and is unlikely to decline in the near future, despite the known and suspected consequences of environmental pollution. Of 33 billion acres of land surface on the earth, approximately three billion acres are cultivated, with 93 percent planted to food crops and most of the rest planted to cotton (Heim 1969). Some of the cropped soils receive a pre-emergence herbicide, fungicide, nematocide, systemic insecticide, postemergent herbicide, one or more postemergent insecticides or fungicides, and a defoliant, all in a single season (Nash 1967). Some pesticides are also used on forested land, and large amounts are used industrially and domestically.

In 1972, the U. S. production of synthetic organic pesticides exceeded 1,157,609,000 pounds, excluding compounds with major nonpesticidal uses. Of this total, herbicides accounted for more than 451 million pounds, insecticides for more than 563 million pounds, and fungicides for almost 143 million pounds (von Rumker *et al.* 1974).

In addition to the intended dispersal of these pesticides onto and into soil and water, considerable amounts must be disposed of without being used: unwanted leftovers, residues remaining in empty containers, and unused stocks of compounds which have been banned. The most common and simplest form of disposal is in soil, and the suitability of soil disposal is obvious for compounds that readily decompose to naturally occurring substances without endangering soil processes, soil microorganisms, or higher organisms in the interim. It is the purpose of this report to evaluate the feasibility of disposing of large quantities of a selected number of pesticides in the soil, but alternative modes of disposal are also considered, since not all compounds lend themselves to soil disposal.

The primary contraindication to soil disposal is undue persistence. Alexander (1965) stressed the principle of microbial fallibility:

> "When the nature of the resistant configuration or backbone is established we can only speculate.......why an organism which does not exist is unable to decompose a compound which does."

Long persistence has, in the past thirty years, led to worldwide contamination by immobile compounds, to poisonings of species other than the intended targets, and to the accumulation of recalcitrant metabolites of greater or lesser toxicity than the parent compound. Therefore, both the mobility and the ramifications of contamination by each pesticide, and/or of its terminal residues, must be considered before large-scale soil disposal is attempted.

This report reviews the published literature on the behavior of 45 pesticides (17 herbicides, 20 insecticides, six fungicides, one fumigant and one acaricide) in soil. For each pesticide, an exhaustive search was made of the literature pertaining to soil persistence and degradation by soil microorganisms, leaching through soil, and volatilization from soil. Because all pesticides on which enough data exist were found to migrate from soil into water, data on persistence, transport, and degradation in water were also included. A thorough search of the literature pertaining to the effects of each pesticide on the non-target microorganisms of soil and water was also made. Data on the toxicity of each compound to other non-target species are included, but these data are reviewed selectively. Particular attention was paid to the hazards of chronic exposure to low levels of pesticides, which would be the most likely consequence of improper disposal, and some data on acute effects are given for comparison. The outline of the review for each pesticide is shown in Figure 1.

Introduction
    Nomenclature, Physical Properties, Synthesis

Degradation
    Biological
    Chemical and Physical
    Photolytic

Transport
    Within Soil
    Between Soil and Water
    Volatilization
    Into Organisms

Persistence

Effects on Non-target Species
    Microorganisms
    Invertebrates
    Plants
    Fish and Amphibians
    Birds
    Mammals

Conclusions

Figure 1

Outline of the review of the literature
for each of 45 pesticides

Sources of the data in this report are: Chemical Abstracts, computer searches, monographs, review articles, and original articles. Each reference contains the source of the data cited. If no additional information is given, the original article was used. If the Chemical Abstracts number is cited, data were taken from this abstract. In the few cases in which a computer search identified an otherwise unascertained article, the name of the program (e.g. TOXLINE) is given as part of the reference. When a review article or monograph was the source of the information, the author of the review is cited. In short, the actual source of the information is cited, as well as the original author when Chemical Abstracts was the source. A list of source references, many of which are not specifically quoted in the text *per se*, follows the references for this section.

Only published literature was reviewed, and theses abstracted in *Thesis Abstracts* were excluded from the definition of published literature. The literature search for each compound began with the patent issue date, and the latest articles cited include several published in 1976, although the search was complete only through September 1975. The last issue of Chemical Abstracts to be scanned was number 13 of Volume 83 (September 29, 1975). Journal searches ended with September 1975 issue in most cases, but exceptions consisted of extended rather than abbreviated searches.

The literature search was exhaustive rather than critical, and many of the studies cited can undoubtedly be criticized. No attempt has been made to evaluate each study in the text, but the conclusions for each compound are based on the excellence as well as on the mass of data supporting them.

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SECTION II CONCLUSIONS

Of the forty-five pesticides reviewed in this report, ten are suitable for soil disposal, twenty-one are unsuitable, and the available data are insufficient for any conclusions in fourteen cases. The feasibility of soil disposal for each pesticide is listed in Table 1.

Since most of the available data deal with relatively small amounts of well-dispersed pesticides and degradation is almost invariably inversely correlated with concentration, bulk disposal of most pesticides will lead to long periods of persistence of the parent compound and/or its degradation products. Transport of persistent compounds through soil is inevitable and transport into ground or surface waters extremely probable. Therefore any large deposits of pesticides in soil must be carefully monitored until decomposition has obviated the possibility of environmental contamination.

In contrast to soil disposal, thermal disposal can present hazards due to formation of toxic gases and other wastes. Decontamination procedures for most of these products of combustion (e.g., chlorine and hydrogen chloride) are well known. While not enough data are available to suggest burning all pesticides, incineration is a rapid and can be a most efficient mode of disposing of many of the pesticides reviewed here.

Table 1. FEASIBILITY OF SOIL DISPOSAL FOR THE 45 PESTICIDES REVIEWED IN THIS REPORT

Herbicide	Soil disposal	Insecticide	Soil disposal	Miscellaneous	Soil disposal
Atrazine	no	Aldrin	no	Captan	yes
Bromacil	?	Chlordane	no	Dicofol	no
CDAA	?	Chlordecone (Kepone)	no	Dodine	?
Chloramben	yes	DDT	no	Maneb	?
2,4-D	yes	Dieldrin	no	Methyl bromide	no
Dicamba	?	Endosulfan	no	Nabam	?
Diquat	no	Endrin	no	Pentachlorophenol	no
Diuron	no	Heptachlor	no	Zineb	?
EPTC	yes	Methoxychlor	yes		
Linuron	no	Mirex	no		
Monolinuron	no	Toxaphene	no		
Monuron	no	Azinphosmethyl	?		
Nitralin	yes	Diazinon	?		
Paraquat	no	Disulfoton	?		
Picloram	no	Malathion	yes		
2,4,5-T	?	Methyl parathion	?		
Trifluralin	yes	Parathion	?		
		Phorate	?		
		Carbaryl	yes		
		Metalkamate (Bux)	yes		

SECTION III HERBICIDES

Atrazine

Atrazine is the common name for 6-chloro-*N*-ethyl-*N'*-(1-methylethyl)-1,3,5-*s*-triazine-2,4-diamine, a pre- and postemergent herbicide introduced by the Geigy Chemical Co. in 1957. It is used for general weed control and for selective weed control in corn, sorghum, sugar cane and pineapple under the trade names AAtrex in the USA and Gesaprim or Primatol in Europe.

Atrazine is a white crystalline solid with a melting point of 173-175°C, a vapor pressure of 3.0 x 10^{-7} mm mercury at 20°C and a water solubility of 33 ppm at 27°C. Its solubility in ethyl acetate is 28,000 ppm; in dimethyl sulfoxide, 183,000 ppm at 27°C. It is synthesized by reacting cyanuric chloride with one equivalent of isopropylamine and one equivalent of ethylamine in the presence of acid acceptors (WSSA 1974). A lengthy review of the triazine herbicides including atrazine was published in *Residue Reviews* 32 (1970).

Degradation—

Biological—The major degradative process undergone by atrazine in soil is its conversion to hydroxyatrazine by loss of the chlorine at the C-6 position (Beynon *et al.* 1972, Harris 1967, Roeth *et al.* 1969, Knuesli *et al.* 1969). Microbial attack reportedly resulted in deethylated atrazine as the major metabolite and deisopropylated atrazine as a minor metabolite (Sirons *et al.* 1973). Decomposition is more rapid in normal than in sterilized soil, and is enhanced by repeated applications of atrazine (Gigineishvili and Dzhugeli 1973). McCormick and Hiltbold

(1966) suggested that atrazine was only incidentally or nonpreferentially involved in microbial metabolism, a situation to which Alexander (1972) has given the name cometabolism. Under laboratory conditions, however, atrazine can serve as a carbon source for *Fusarium roseum* (Couch et al. 1965), *Rhizopus stolonifer* and several species of *Aspergillus* and *Fusarium* (Kaufman and Blake 1970) and for *Mucor spinosum*, *Aspergillus nidulans* and *Penicillium tardum* (Nepomiluev and Kuzyakina 1972). Also under laboratory conditions, fungi could be adapted to use atrazine as a nitrogen source (Manorik and Malichenko 1971), giving half the rate of growth of controls in cultures of *Cephalosporium acremonium*, *Cladosporium herbarum*, and in several *Penicillium* and *Aspergillus* species. Atrazine did not, however, suffice as a carbon source in this study (Mickovski and Verona 1967). Soil bacteria decomposed 70 percent of an unspecified level of atrazine in 18 days, with *Bacillus megaterium* and *B. aglomeratus* identified as most active (Voinova and Bakalivanov 1970).

The degradation of atrazine by soil fungi is restricted to the side chains, and possibly only to laboratory cultures (Kaufman and Blake 1970). When ring-labeled atrazine was incubated in anaerobic (flooded) organic soil, only 0.02 to 0.59 percent of the radioactive label was converted to carbon dioxide. Addition of starch or ammonium sulfate increased the $^{14}CO_2$ evolution somewhat, as did aeration of the soil (Goswami and Green 1971). When chain-labeled atrazine was incubated in soil at 25°C and 80 percent of field moisture capacity, only one percent of the radioactive label was converted to carbon dioxide. At lower temperatures or in less moist soils, even less $^{14}CO_2$ was evolved (Roeth et al. 1969).

In cultures of 21 planktonic algae, atrazine was not removed from water by the algae (Butler et al. 1975). Degradation in soil followed first-order kinetics with no lag period (Zimdahl et al. 1970).

In fish, exposure to ten ppm atrazine did not result in significant accumulation in muscle tissue, and the atrazine was rapidly degraded (Bathe et al. 1972). Dairy cows did not excrete atrazine in their milk, but two percent was excreted in the urine as unaltered atrazine. No metabolites were identified (St. John et al. 1964). Paulson (1975) in his review of the animal metabolism of atrazine, stated that 6-chlorotriazines are eliminated more slowly than 6-methoxy- or 6-methylthiotriazines, and that dealkylation is inversely related to the length of the side chain. No ring cleavage has been reported in animals, and almost none of the many metabolites reported have been identified. In rat liver *in vitro*, dealkylation was the primary mode of degradation and no dechlorination occurred (Dauterman and Muecke 1974).

Photolytic--Atrazine in aqueous solution was converted to hydroxyatrazine by ultraviolet light at 253.7 nm (Pape and Zabik 1970), a reaction which also occurred in natural sunlight (Pape and Zabik 1972).

Chemical and physical--The nonenzymatic decomposition of atrazine in soil is unlikely under anaerobic conditions in the absence of adsorption (Hance 1967) but feasible if adsorption occurs (Hance 1969). Nonenzymatic hydrolysis was considered the major mechanism of atrazine detoxification in some studies (Best and Weber 1974, Zimdahl et al. 1970). In comparisons between sterilized and unsterilized soil, however, only 13.6 percent of atrazine was decomposed after one month in sterile soil, but 23 percent in unsterilized soil. The differences were even more marked when repeated applications of atrazine were made to the soils (Giginetshvili and Dzhugeli 1973). Brown and White (1969) found atrazine to be less susceptible to hydrolysis than trietazine, chlorazine, propazine, ipazine or simazine, although the observed hydrolysis was relatively great since atrazine was not as heavily adsorbed by the clay. Armstrong and Chesters (1968) did not consider adsorption hydrolysis common, and also argued against the nonbiological hydrolysis of atrazine as a major method of degradation under normal conditions. Among the factors favoring atrazine degradation under environmental conditions were increasing temperatures (Roeth et al. 1969, McCormick and Hiltbold

1966).

Paris and Lewis (1973) summarized the welter of data by noting that dealkylation does not destroy the phytotoxicity of atrazine, whereas hydrolysis does; the former is presumed to be biological and the latter nonbiological.

Concentrations of 50 to 100 mg/liter of atrazine were decomposed by 20 to 30 minute contact with five to 20 mg of chlorinated lime (Martynuk and Gshegotskii 1967). Metallic sodium and liquid ammonia decomposed atrazine 100 percent, while substituting lithium for sodium resulted in 17.5 percent intact atrazine (Kennedy *et al.* 1972a). Atrazine could also be partially decomposed by 15N ammonium hydroxide (Kennedy *et al.* 1972b).

When atrazine was heated in a muffle furnace, 87.8 percent was decomposed at 600°C, and 89 percent at 1000°C (Kennedy *et al.* 1969). The volatile products of burning atrazine at 900°C were carbon monoxide, carbon dioxide, hydrochloric acid and ammonia (Kennedy *et al.* 1972a, 1972b). Some decomposition occurred at temperatures as low as 250°C, turning atrazine into yellow flakes which were tentatively identified as primary or secondary amines (Stojanovic *et al.* 1972b). Tadik and Ries (1971) identified the decomposition of atrazine between 240°C and 250°C to consist of elimination of the isopropylamino and ethylamino chains, possibly with a cyclic transition state. They also decomposed triazine herbicides by sonication, but did not conclusively identify the products.

Transport:

Within soil--Transport of atrazine is inversely correlated with adsorption of the herbicide to soil and soil constituents (Rodgers 1968). Several attempts have been made to fit the data for atrazine adsorption to theoretical or empirical models (Hayes *et al.* 1968, Hance 1969b, Guth 1972, Bailey and White 1972, Huggenberger *et al.* 1973). Although qualitative fit was achieved, the models were not considered good predictors of atrazine behavior in the field (Huggenberger et al. 1973, Crawford and Donigan 1973).

The major factors influencing the adsorption of atrazine on soil are the soil composition, soil moisture, pH, and temperature. Atrazine is heavily adsorbed on organic matter in soil (Hilton and Yuen 1963) or activated charcoal (Jordan and Smith 1971). In the laboratory it has been shown atrazine binds to the insoluble lignin or humic acid fractions of soil, with only minor binding associated with polysaccharide components and virtually none with ethanol-soluble components (Dunigan and McIntosh 1971). Plant residues, fungal resting structures, fecal pellets and animal cysts adsorbed atrazine heavily (Grossbard et al. 1970). Aliphatic hydroxyl groups, aliphatic carboxyl groups and phenolic hydroxyl groups are thought to be active in the soil binding process (Turski and Steinbrich 1972).

Adsorption of atrazine on charcoal increased its persistence in soil, as did addition of clay to the soil (Moyer et al. 1972a) but Hance (1974) argued that adsorption of atrazine by soil organic matter did not affect degradation. Weber (1970b) noted that reversible adsorption could lead to increased, delayed or decreased phytotoxicity. The relative adsorption of atrazine on different soils was: marsh > fine grain > light, sandy soil (Gorzelak 1972); silt or silty clay > sandy loam (Harris 1966); chernozem > Kraznozem > sod podzol (Khlebnikova 1974).

On clay, atrazine adsorption is dependent both on pH and the cation exchange capacity of the clay (Hance 1969a). Adsorption on montmorillonite, but not on peat, was reversible even when the adsorbent was dried and re-wet (Moyer et al. 1972b), and adsorption on kaolinite was fully reversible (Scott and Lutz 1971). Under some experimental conditions, atrazine is not adsorbed by kaolinite (Talbert and Fletchall 1965).

At pH less than six, increasing the temperature resulted in both increased binding and in increased recovery of atrazine when temperature varied: from 85 percent recovery at 0.5°C to 98 percent recovery at 40°C, while recovery at pH 7 was only 69 percent (McGlamery and Slife 1966). Adsorption of atrazine usually decreased as pH increased above pH 7 (Hance 1969a, Weber 1970a, Nearpass 1967, Green and Corey 1971, Colbert et al. 1975) but two silt loam soils with natural pH 8.5 and 9.6 adsorbed atrazine very well (Colbert et al. 1975).

Increasing the temperature from three to 25°C produced a four-fold increase in the binding of atrazine to humic acid (Li and Felbeck 1972) and temperature affected atrazine adsorption on bentonite but not on muck (Harris and Warren 1974). Talbert and Fletchall (1965), however, reported decreased adsorption of atrazine at higher temperatures.

When the soil to water ratio was soil : water :: 1 : 10, adsorption of atrazine on soil was five times as great as when the ratios were soil : water :: 3 : 1. The data suggested that the size of the soil aggregates affected adsorption (Grover and Hance 1970).

The relative adsorption of herbicides on soil was: 2,4-D < fluometron < prometone < simazine, atrazine, diphenamid < prometryne << chlorpropham << trifluralin << benefin (Scott and Phillips 1972) and, among triazines: propazine < atrazine < simazine < prometone < prometryne (Talbert and Fletchall 1965). On a six point scale of soil mobility, atrazine was rated at 2.7 in silty clay loam and as 3.5 in sandy loam, which is considered moderately mobile (Harris 1967). Among the triazines, the chlorotriazines are less mobile than the analogous methoxytriazines but more mobile than the *S*-methyl triazines (Helling et al. 1971).

The relative leaching of seven *s*-triazines was: atratone > propazine > atrazine > simazine > ipazine > ametryne > prometryne (Rodgers 1968). Increasing soil bulk inhibits the diffusion of atrazine through soil, while high soil moisture enhances it (Ritter et al. 1973, Lavy 1970). Granular atrazine leached further if formulated on ammonium sulfate than on limestone (Gast 1962). Agricultural soils have a greater than necessary capacity for binding agricultural levels of atrazine (Snelling et al. 1969), but the precise binding capacity depends on the state of decomposition of the organic matter as well as on its amount (Walker and Crawford 1968). Electrolytes can also alter the adsorptive capacity of soil (Hurle and Freed 1972).

Studies on the leaching of atrazine in soil columns and under field conditions demonstrate the variability of soil penetrance. In quartz sand in 30 cm soil columns, atrazine penetrated no more than ten cm when ten cm water were used for leaching, and no more than 20 cm if the sand was leached with 20 cm of water (Vega 1974). In soil columns leached with three cm water per day, atrazine penetrated to 24 inches (60 cm) with 18 inches of water in silt loam and in sandy loam, but only to 20 inches (50 cm) in loam. With three inches (7.5 cm) water, phytotoxic amounts of atrazine leached to three or four inches (≤ 10 cm) in loam, six inches (15 cm) in sandy loam, and eight to ten inches (≤ 25 cm) in silt loam (Ivey and Andrews 1965).

When applied to the top two cm of chernozem and podzolic sandy loam, atrazine penetrated seven to eight cm in the former, but more than 13 cm in the latter within six months (Krzecki and Kowalski 1976) while four to six kg/ha penetrated light chestnut soils to ten cm (Stepanova 1965). Ten to 12 kg/ha of atrazine leached to 20 cm in tea plantations, but 12 and 16 kg/ha penetrated 30 cm in orchard soils (Tabagua 1975).

In chernozem-smolnitsa soil, 1.5 kg/ha of atrazine was most phytotoxic in the four to six cm layer for two to three months, and in the ten to 12 cm layer thereafter until it decomposed (Andreeva-Fetvadzieva 1967).

In moist subtropical soils, atrazine leached only 40 cm during 1.5 to two months in waterlogged soil, but 50 to 70 cm in red soil (Spiridonov et al. 1970b). At temperatures below ten^{0}C, five to 20 kg/ha persisted long enough to leach to 70 cm, but at 20^{0}C, more rapid decomposition decreased the penetration to 30 cm (Spiridonov and Yakovlev 1967). Repeating atrazine treatment had neither increased nor decreased the degree of penetration after three annual applications (Spiridonov *et al*. 1970a). In red soils under subtropical conditions, atrazine penetrated 50 cm within forty days after application of ten kg/ha, and to 70 cm 40 days after 20 kg/ha were applied (Spiridonov *et al*. 1968). Khubutiya and Giginelshvili (1971) detected residues from 16 kg/ha or less of atrazine at 40 cm in brown forest soils within 60 days. After 25.0 cm precipitation spread over a winter, atrazine had leached to 20 cm in loam, but to 80 cm in sand (Pop *et al*. 1968) whereas White and co-workers (1967) recorded only three inch penetration (7.5 cm) after 2.5 inches (6.3 cm) of simulated rain. Bailey and White concluded that the depth of pesticide penetration into soil depends not only on the degree of reversible and irreversible absorption, but also on chemical reactions of the pesticide, its solubility, the amount applied, and on the average flow velocities, the pore geometry of the soil, and the soil moisture at the time of application. Degradation was suggested as a major factor determining the penetration of atrazine into the soil (Green *et al*. 1968, Harris *et al*. 1969).

In assessing the information available on the dissipation of atrazine in the soil, and its effects on the environment during such dissipation, two elements must be stressed: first, atrazine is by function a soil treatment whose action depends on its persistence and most studies dealt with the herbicidal persistence of atrazine and its phytotoxic metabolites in the plow layer and/or root growth layer of the soil; secondly, application of atrazine to the soil was in amounts and practices which might approximate agricultural usage, essentially all of the atrazine so applied is accessible to soil, if not to air, sun, and plants. It was estimated that the half-life of atrazine at 20°C in sterile soil would be 9 to 116 years (Hance 1967). The estimate, extrapolated from data of herbicide slurries at 85-107°C, was criticized because adsorption is an exothermic process which would be decreased at the high temperatures of the slurries (Armstrong and Chesters 1968). What the consequences of preventing adsorption would be has not been considered, but virtually all data point to greater biological persistence, and/or greater leaching, in light soils with little adsorption.

Between soil and water--On thin-layer chromatography, atrazine was found to be more mobile than its metabolites, and to migrate independently of them. Under laboratory conditions, water transport at 25°C was 15.3×10^{-8} cm^2/second (Helling 1971). When fallow soil was treated with atrazine, a 2.5 inch simulated rainfall caused the largest loss of atrazine soon after application, and the largest losses in such a rain occurred in the first portion of the rainfall. The amount depended on the soil temprrature, but was of the order of 2.3 ppm in the runoff when 2.2 kg/ha of atrazine had been applied (White *et al*. 1967). When rain fell after seven days, about 0.15 percent of the atrazine was lost in runoff from contour-planted soil; ridge-planting reduced the runoff and consequently the atrazine losses.

Of the atrazine lost in runoff, proportionally more was sediment-associated than water-associated (Ritter *et al*. 1974). In a heavy-texture soil, four to six times as much sediment was lost from conventionally tilled plots as from no-till plots, and surface water contained more atrazine than did the water in drainage tiles (Schwab *et al*. 1973). Atrazine polluted groundwater in wet years and in the spring in which it was applied (Leh 1968).

In an aquifer, when 1.28 ppm of several pesticides were introduced into a dual function well, atrazine migrated 30 feet from the point of entry after 50 to 150 hours, but never more than 150 feet during the ten days before the aquifer was decontaminated (Schneider *et al*. 1971). After application in fish ponds, water residues decreased 90 percent in two weeks, and atrazine levels in sludge exceeded water levels within days. After eight weeks, water levels were at approximately one percent of the original levels (Maier-Bode 1972). Another indication of the contamination of water by atrazine is the occurrence of atrazine and its metabolite in the drinking water of New Orleans (Anonymous 1974b).

Volatilization--Approximately 80 percent of the atrazine on aluminum planchets volatilized within 48 hours, but the addition of even small amounts of co-extracted plant materials reduced volatility sharply (Walker 1972). Under infra-red lamps at 52° to 61°C, 95 percent volatilized from planchets in 24 hours (Foy 1963) but only 11 percent evaporated from the soil surface in two days at 40°C at an air velocity of two liters/minute. Decreasing the temperature or the air velocity significantly reduced losses due to volatilization (Burt 1974). More atrazine volatilized from wet sand than from dry (Kearney *et al*. 1964). Jordan *et al*. (1970) reported a 65 to 85 percent loss of phytotoxicity in 60 days when atrazine was exposed to light, but the effects of photolysis and volatilization were not separated.

Into organisms--Pond treatment with atrazine led to accumulation of the herbicide by algae and by water fleas (*Daphnia*), but carp, although they also accumulated atrazine, eliminated it rapidly (Maier-Bode 1972). In the terrestrial-aquatic model ecosystem (Metcalf *et al*. 1971), the ecological magnification values (EM) for atrazine in the alga, snail and fish were only 75.6, 7.5 and 11.0, respectively. These organisms also contained two dealkylated metabolites of atrazine which had EM values similar to those of atrazine itself. Atrazine was judged unlikely to be heavily accumulated in aquatic food chains (Sanborn 1974, Metcalf and Sanborn 1973). When laying hens were fed 0.5 ppm atrazine for six weeks, no residues were detected in the yolks of their eggs (Foster *et al*. 1972).

Persistence--

Atrazine was less persistent than either amiben or 2,3,6-TBA (Burnside 1965). Bryant and Andrews (1967) stated that atrazine phytotoxicity was lost after 16 weeks in clay loam and after 24 weeks in silt loam, but considerable experience suggests that phytotoxicity can carry over to the next season (WSSA 1974). Andreeva-Fetsadshieva (1967) stated that atrazine phytotoxicity persisted for 4.5 months in chernozem-smolnitsa soil when 627 cm of rain fell, but persisted longer during droughts. In a Nebraska loam, atrazine phytotoxicity from treatment with ten lbs/A (11.2 kg/ha) persisted into the third growing season (Burnside *et al*. 1965) and similar results were observed in the USSR from 20 kg/ha (Arkharova 1973). Persistence increased as concentration increased (Hance and McKone 1971) but decreased as temperatures increased (Roeth *et al*. 1969) and as humidity above anaerobic soils approached 60 percent (Spiridonov and Kamenskii 1970).

The climate did not affect atrazine persistence as much as the soil characteristics, but a drier climate led to greater persistence than a wet climate. Coarse soil led to greater atrazine carryover between seasons than did fine soil (Burnside *et al*. 1969). In Austria, phytotoxic persistence increased with increasing soil humus in dry weather but decreased with increasing soil humus in wet weather (Neururer 1972). Clay or charcoal added to soil decreased phytotoxic persistence of atrazine (Harvey 1973, Moyer *et al*. 1972a) and persistence was greater in silty clay loam than in loam (Burnside *et al*. 1971).

In silty loam, atrazine persisted longer at 90 cm than at 60 cm, and the lowest persistence occurred at 15 cm (Lavy *et al*. 1973). (See Table 7). When placed in tubes at three, nine and 15 inches in the soil at twelve locations in the United States and Puerto Rico, persistence was 61 percent greater after three months at 15 inches (37.5 cm) than at three inches (7.5 cm). Geographical variations in persistence were not entirely explainable (Harris *et al*. 1969).

In Puerto Rican sugarcane soil, atrazine was more persistent than diuron (Liu *et al*. 1971). Under field conditions the relative persistence of six herbicides was: simazine and diuron > trifluralin and atrazine > fluometron > prometryne (Horowitz 1969). Atrazine was also found in 14.1 percent of 199 soils with a history of atrazine usage (Wiersma *et al*. 1972).

Table 1. PERSISTENCE OF ATRAZINE IN SOIL

% residue	# days	% lost/day	Conditions	Reference
>0	1230	<0.08	90 cm soil incorporation	Lavy *et al.* 1970
>0	510	<0.20	40 cm " "	Ibid.
>0	425	<0.24	phytotoxic persistence	Isom *et al.* 1970
>0	150	<0.67	15 cm soil incorporation	Lavy *et al.* 1970
>0	150	<0.67		Heinisch 1973
33	84	0.80	10 ppm on Norfolk clay loam	McCormick and Hiltbold 1966
30	84	0.83	10 ppm on Decatur clay loam	Ibid.
≥70	34	≤0.88	1-100 ppm, Hawaiian soils	Roeth *et al.* 1969

Effects on Non-Target Species-

Microorganisms--Of the numerous studies on the effects of atrazine on soil respiration, only one showed an effect at less than 20 kg/ha: Spiridonov and Spiridonova (1971) claimed five to ten kg/ha of atrazine stimulated soil respiration while 20 kg/ha depressed it. Depression of soil respiration was also observed by Whitworth et al. (1965), Liu and Cibes-Viade (1972), Eno (1962), Bartha and Lanzilotta (1967), but in all cases more than 160 kg/ha were applied. Stimulation of soil respiration for 30 days at over 1000 kg/ha was observed by Bartha and Lanzilotta (1967); inhibition occurred after the 30 day period. Atrazine also stimulated soil respiration at levels not exceeding 25 kg/ha (Spiridonov and Spiridonova 1971, Liu and Cibes-Viade 1972, Balicka and Sobieszczanski 1969). Most common, however, were reports of no effect (Liu and Cibes-Viade 1972, Kulinska 1967a, Eglite and Folkmane 1967, Corke and Robinson 1960) at levels up to 512 ppm (1138 kg/ha) (Eno 1962). In an experiment specifically designed to guage the effects of soil disposal of pesticides, Stojanovic et al. (1972a) concluded that five tons/A of pure atrazine (5,000 ppm) inhibited soil respiration by 22 percent over a 56 day period, while formulated atrazine inhibited soil respiration by 43 percent over 56 days. These data fit the theory that, whereas agricultural levels of atrazine do not inhibit soil respiration, massive amounts do.

At levels approximating its agricultural use, atrazine almost always stimulated nitrification and soil nitrate levels (Zavarzin 1966, Tulabaev and Tamikaev 1967, 1968, Tsvetkova 1966, Belobrov 1972, 1974, Andreeva-Fetvadzhieva and Kolcheva 1970), although Siradze (1969) noted that the stimulation of nitrification occurred the second and third growing season after atrazine application. Stimulation of nitrification was not observed above 15 kg/ha, but Eno (1962) noted no significant effect of atrazine at levels below 512 ppm (1,138 kg/ha) with inhibition at higher levels. Casely and Luckwill (1964) and Thornsburg and Tweedy (1973) observed no significant effects on nitrification at levels of 12.6 kg/ha and 26.7 to 53 kg/ha, respectively.

Four kg/ha stimulated ammonification in the second and third season after atrazine was applied (Siradze 1969), but Zavarzin and Belyaeva (1966) reported that unspecified levels of atrazine inhibited ammonification. The available potassium and phosphate levels in the soil were increased in podzol (Tsvetkova 1966) and in Ukrainian forest steppes treated with six to ten kg/ha (Belobrov 1972) but not in chernozem treated with six to ten kg/ha (Belobrov 1974). Four kg/ha caused increases in soil phosphate levels in the second and third season after atrazine treatment (Siradze 1969).

Atrazine increased the total numbers of soil microorganisms at three kg/ha in light chestnut soil (Stepanova 1969) and at five to ten kg/ha (Spiridonov and Spiridonova 1971), but higher levels were inhibiting (Spiridonov and Spiridonova 1971, Kuz'mina 1968). In peat-podzolic soil, however, 75 kg/ha stimulated the main groups of microorganisms (Mashtakov et al. 1962). The effect observed depended on time of observation, since an immediate decrease in the total number of microorganisms in light chestnut soils was followed, after two months, by an increase to twice the numbers in control soil (Stepanova 1967). Pantera (1972) considered atrazine to be less potent an inhibitor of soil microorganisms than monuron, but more potent than either prometryne or Antipers. Zubets (1970) considered atrazine to be a strong microbial inhibitor. At 100 ppm, atrazine did not affect microflora in Macedonian soils (Mickovski 1968).

The effects of atrazine on nitrogen-fixing bacteria vary from inhibition (Rankov 1968, Wegrzyn 1971, Tulabaev 1971, Zubets 1973) through no significant effects (Peshakov et al. 1969, Casely and Luckwill 1964) to stimulation (Zubets 1970, Spiridonov and Spiridonova 1971, Andreeva-Fetvadzhieva and Kolcheva 1970). Although higher levels tended to be inhibitory and lower levels stimulatory, no conclusions could be drawn as to absolute values to achieve either.

Atrazine inhibited ammonifying bacteria under unspecified conditions in soil (Peshakov et al. 1969), at three kg/ha on meadow soil (Tulabaev and Tamikaev 1968), and cellulolytic bacteria were probably inhibited in light soils (Klyuchnikov et al. 1964, Stepanova 1967) although no significant effect was reported in one study (Amantaev et al. 1963). Stimulation of cellulolytic bacteria occurred on peat-bog soil (Gurinovich et al. 1962) and at five to 20 kg/ha (Spiridonov and Yakovlev 1968, Spiridonov and Spiridonova 1971). Bacterial cellulolytes were more sensitive to atrazine than cellulolytic fungi, and no resistance was observed in cultures from atrazine-treated soil (Sobieszanski 1968). Effects on cellulolytic microorganisms were not, however, as severe as effects on nitrifying bacteria (Tulabaev 1971).

Azotobacter was inhibited by unspecified levels of atrazine (Peshakov et al. 1969) and by three kg/ha (Tulabaev 1971) but not by one ppm or ten ppm in cultures (Wegrzyn 1971). *Bacillus subtilis* was inhibited in cultures containing unspecified levels of atrazine (Kuz'mina 1968) and *Coccaceae* increased relative to *Bacillaceae* (Ghinea 1967) but no effect on *Arthrobacter* was seen in the presence of two to five percent (20,000 to 50,000 ppm) atrazine if sugar was added to the culture (Micev and Bubalov 1972).

Inhibition of soil fungi was reported repeatedly (Bakalivanov 1971, 1972, Klyuchnikov et al. 1964, Spiridonov and Spiridonova 1971, Zubets 1973, Simon-Sylvestre 1974) as was no effect (Peshakov et al. 1969, Amantaev et al. 1963, Tulabaev and Tamikaev 1968, Belobrov 1972). Stimulation of fungi was also reported at two or 12 kg/ha in sod-podzolic soil (Zubets 1973) and at about nine to 18 kg/ha (Kuzyakina 1971) and occasionally for low levels of atrazine in cultures (Manturovskaya 1970). At low levels, saprophytic fungi were stimulated more than pathogenic fungi while at higher levels (17.5 ppm) all species were inhibited (Richardson 1970).

When five tons per acre of atrazine (5,000 ppm) were incorporated into loam soil, a slight stimulation of fungi occurred. *Streptomyces* were stimulated by analytical grade atrazine but inhibited slightly by formulated atrazine (80 percent wettable powder) (Stojanovic et al. 1972a). Actinomycetes were stimulated by ten kg/ha in moor soil (Kuz'mina 1968) and their numbers unaffected by two or 12 kg/ha in sod-podzol (Zubets 1973) but their taxonomy was altered in the absence of numerical effects (Ghinea 1967). *Trichoderma* growth was inhibited for ten months on peat-bog soil (Gurinovich et al. 1962) and even 0.01 kg/ha depressed growth of *Fusarium*, but not of *Penicillium* or *Aspergillus*, on turf-podzol (Gorlenko et al. 1969). Atrazine more drastically affected fungi than either simazine or prometryne (Manturovskaya 1970).

Voets, Meerschman and Verstraete (1974) analysed the effects of 15 years treatment of orchard soil with atrazine, at four kg/hg/year. Changes in microbial population included temporary increases in the numbers of ammonifying and proteolytic organisms, and temporary decreases in the numbers of nitrifying, amylolytic and denitrifying organisms. The numbers of *Azotobacter* increased permanently, and permanent decreases occurred in the numbers of anaerobic, spore-forming, and cellulolytic microorganisms. There was a concomitant loss of ground cover and soil organic matter.

Atrazine inhibited soil algae (Mikhailova and Kruglov 1973), particularly at high levels (Pantera 1970), but was less toxic than ametryne, neburon, or diuron (Hollister and Walsh 1973). Pillay and Tchan (1972) ranked the relative toxicity of six herbicides to algae as: diuron >> neburon > monuron > atrazine > simazine > atratone. Five ppm atrazine caused 75 percent inhibition of *Chlorococcum* in culture, and 0.5 ppm caused 70 percent inhibition of *Chlorella*. Even 70 ppm did not totally inhibit the growth of these algae, however (Virmani et al. 1975). Pidgaiko (1971) considered atrazine toxic to zooplankton reproduction, but the level of treatment was not available.

Besides differences in rates of application, soils, species examined, and types of measurements made, the experiments cited are heterogeneous with respect to the length of time of observation. As noted by Stepanova (1967), the application of herbicide may alter conditions by altering moisture retention and the amount of organic matter present on or in the soil. The effects would differ in preemergence weed control in a plowed field from general weed control in an orchard or fallow field. In the first case, atrazine would tend to minimize the amount of organic material accumulating in or on the soil, and maximize moisture loss. In the latter cases, decaying organic matter would be increased by herbicide treatment. The responses of soil microorganisms might well differ. It is known that some molds and some bacteria can utilize atrazine as a nitrogen source and, to a lesser extent, as a carbon source (Goswami *et al.* 1971, Nepomiluev and Kuzyakina 1972, Mickovski and Verona 1967, and Kaiser *et al.* 1970). Manorik and Malichenko (1971) suggested that there is adaptation by soil organisms to the presence of triazines, a phenomenon which could explain shifts in overall soil respiration as well as increases in particular species.

Plants--In addition to its herbicidal effects, atrazine is mutagenic in barley (*Hordeum vulgare*) (Wuu and Grant 1966, Sharma 1973) although Stroev (1968) did not induce mutations in barley at 12 hours' exposure to less than 10^{-3}M concentrations of atrazine. A metabolite of atrazine produced by atrazine-treated corn (*Zea mays*) is mutagenic in both corn and yeasts (Galston 1976, Gentile and Plewa 1976, Plewa and Gentile 1975, 1976a, 1976b, 1976c).

Invertebrates--At levels greater than 19 ppm, atrazine synergized parathion toxicity to fruit flies or mosquitos (*Aedes aegypti*) (Liang and Lichtenstein 1974). In grassland soil, agricultural levels of atrazine decreased the numbers of wireworms, springtails and earthworms, but not of millipedes or mites. The effect may have been secondary to changes in vegation (Fox 1964). Numerous species of aquatic invertebrates were reduced by atrazine treatments of 0.5 to two ppm, but water beetles and damselfly nymphs increased (Pimentel 1971). One mg/ml (1000 ppm) increased fertility in the first generation of water fleas (*Scapholeberis mucronata* and *Ceriodaphnia quadrangula*) in the first generation but decreased fertility in subsequent generations (Shcherban 1972).

Fish and amphibians--Schluster (1970) considered atrazine toxic if used carelessly near water, but not if carefully used. Darwazeh and Mulla (1974) noted no toxicity of 10 ppm atrazine to mosquito fish (*Gambusia affinis*) after 48 hours, but one pound per acre (1.12 kg/ha) killed 26 percent of the fish after four days. Hiltibran (1967) reported that bluegill fry and green sunfish fry survived eight to ten days' exposure to ten ppm of atrazine. The 48-hour LC_{50} for rainbow trout was 12.6 ppm (Pimentel 1971).

Birds--The LC_{50} of atrazine for two week old quail, pheasants, bobwhites and mallards was above 5,000 ppm, but seven percent of the quail and 30 percent of the mallards died when fed 5,000 ppm for three days (Heath et al. 1972). The hatching rate of hens' eggs was not reduced by 100 ppm atrazine injected into the yolk sac, and was reduced by only ten percent when 200 ppm were injected (Dunachie and Fletcher 1967, 1970).

Mammals--The acute oral LD_{50} of atrazine in rats is 3,000 to 3,080 mg/kg (Pimentel 1971, Bashmurin 1974). Atrazine caused bone marrow inhibition and chromosomal aberrations when 0.033 to 0.1 x LD_{50} was injected intraperitoneally into rats (Kulakov 1970) and morphological changes leading to death at 20 mg/kg/day in rats (Nezefi 1971).

Conclusions-

Neither the persistence of atrazine itself nor its effects on soil microorganisms is extreme. Nevertheless, there is no conclusive evidence for degradation of the triazine ring under environmental conditions. Atrazine and one of its metabolites have been detected in the New Orleans water supply, suggesting widespread pollution and/or considerable mobility of these compounds. Large-scale soil disposal of an already pervasive contaminant of uncertain biodegradability cannot be recommended.

Bromacil

Bromacil is the common name for 5-bromo-3-*sec*-butyl-6-methyluracil, a general herbicide introduced by E. I. DuPont de Nemours Chemical Co. in 1962 as Hyvar, and also marketed by U.S. Borax Co. as Hibor. Bromacil is a white, odorless, crystalline solid with a melting point of 158° to 159°C and a water solubility of 815 ppm at 25°C. Its solubility in ethanol is 134,000 ppm at 25°C. Formulations include granules and liquids, alone or in combination with other herbicides. A review of the toxicology, environmental fate, chemistry, production and use of bromacil has recently been published (Burnam et al. 1975).

Degradation—

Biological—Wolf and Martin (1974) applied 2.88 ppm of 2-^{14}C-bromacil to a neutral sandy loam soil. After 115 days, 53 percent of the bromine content of the soil was still present as bromacil in one study, but 22.1 percent of the ^{14}C was released as $^{14}CO_2$ over a 600 day observation period if soil water-holding capacity was maintained at 60 percent. When soil water was at 100 percent of capacity, less than 0.5 percent of the ^{14}C evolved as $^{14}CO_2$ in 145 days but all the bromacil had been altered to some extent. Gardiner et al. (1969b) reported that 25.3 percent of ^{14}C-bromacil decomposed to $^{14}CO_2$ after nine weeks in the laboratory, but not in the field. Metabolites included 5-bromo-3-*sec*-butyl-6-hydroxymethyluracil; 5-bromo-3-(2-hydroxy-1-methylpropyl)-6-methyluracil; and 5-bromo-3-(3-hydroxy-1-methylpropyl)-6-methyluracil. The mutagen 5-bromouracil was not found. Zimdahl and co-workers (1970) characterized the degradation of bromacil in soil as following first-order kinetics with no preliminary lag phase. No data on specific microorganisms which degrade bromacil were available.

Rats fed 1,250 ppm bromacil for one month metabolized it to 5-bromo-3-*sec*-butyl-6-hydroxymethyluracil which was excreted in their urine.

mostly in conjugated form. Five or six other metabolites, but not 5-bromouracil, were also found (Gardiner *et al.* 1969a). Dairy cows fed five ppm of bromacil excreted 0.019 ppm in their milk; excretion increased to 0.13 ppm when 30 ppm bromacil were fed. No urinary or fecal bromacil was found, but no attempt was made to identify metabolites (Gutenmann and Lisk 1970).

Photolytic--When one to ten ppm of bromacil in aqueous solution were exposed to summer sunlight for four months, very little was converted to 5-bromo-6-methyluracil. Recovery of bromacil was almost quantitative (Moilanen and Crosby 1974).

Chemical and physical--Liquid ammonia and metallic sodium decomposed 100 percent of analytical grade bromacil (Kennedy *et al.* 1972a) but 18*N* H_2SO_4 only partially decomposed it (Kennedy *et al.* 1972b). At 250^{0} C, bromacil was changed from a white to a black solid; the major decomposition product was tentatively identified as 3-*sec*-butyl-methyl-uracil (Stojanovic *et al.* 1972b). When bromacil was heated to 600^{0}C in a muffle furnace, 88.6 percent of the formulated herbicide (80 percent wettable powder) was lost; at 1000^{0}C, 91.3 percent was lost (Kennedy *et al.* 1969). The volatile products of burning bromacil at 900^{0}C were carbon monoxide, carbon dioxide, chlorine, hydrogen chloride and NH_3. Hydrogen bromide was not produced (Kennedy *et al.* 1972a).

Transport-

Adsorption of bromacil to soil corresponds to a Freundlich isotherm, suggesting physical rather than chemical adsorption (Rhodes *et al.* 1970, Hague and Coshow 1971). After six to seven years' application of bromacil to loam orchard soil, less than three percent of the bromacil was recovered at depths of one meter although considerable leaching to the 10-20 cm layer had occurred. Leistra *et al.* (1975) considered this less leaching than was implied by the mobility of bromacil on thin-layer chromatographs.

Persistence-

Bromacil was more persistent in sandy than in clay soil, and more persistent in lower soil layers than in surface layers (Stecko 1971). One year after the last of six to seven annual applications of bromacil to loam soil at 1.5 to 3.0 kg/ha/year, 54 percent of an annual dose was present in the soil. Leistra and co-workers (1975) calculated this to correspond to a loss of 35 percent per year. The persistence in soil was even greater than these data suggest, since 20 to 30 percent of the bromacil could have been taken up by plants (Leistra _et al._ 1975). In California agricultural soils bromacil persisted for over 30 months (Lange _et al._ 1968). Weber and Best (1972) estimated that full weed control by bromacil persisted for over 18 weeks, and a half-life of five to six months was estimated by Gardiner _et al._ (1969) in silt loam when four lbs/A (4.48 kg/ha) were applied. Persistence in the laboratory was much shorter as 25.3 percent of the bromacil decomposed to CO_2 after nine weeks (Gardiner _et al._ 1969).

Effects on Non-Target Species-

Microorganisms--In the laboratory, 2.88 ppm bromacil did not alter carbon dioxide evolution in neutral sandy loam (Wolf and Martin 1974) nor did four ppm in sandy loam (Steyn and Wolff 1969) or 100 ppm in two clay loams or in sandy loam (Liu and Cibes-Viade 1972).

When pure bromacil was incorporated into loam soil at five tons per acre (5000 ppm), 16 percent inhibition of carbon dioxide evolution occurred, but formulated bromacil inhibited carbon dioxide evolution by only nine percent. Bacteria were stimulated by pure and by formulated bromacil at 5000 ppm, fungi were stimulated by 5000 ppm pure bromacil, and streptomycetes by 5000 ppm of either pure or formulated bromacil (Stojanovic _et al._ 1972a). Algal growth, in contrast, was strongly inhibited by 0.32 ppm of bromacil (Pillay and Tchan 1972), a result not surprising for an inhibitor of photosynthesis. Bromacil was not mutagenic in a bacteriophage, and did not inhibit the mutagenicity of 5-bromouracil (McGahen and Hoffmann 1966).

The median tolerance limit for bluegills and rainbow trout was about 100 ppm at 24 hours, falling to 75 or 80 ppm at 48 hours (WSSA 1974). The eight-day LC_{50} for bobwhite quail and for mallard ducklings was over 10,000 ppm, and the acute oral LD_{50} for rats was 5200 mg/kg (WSSA 1974).

Conclusions:

The few data on bromacil transport and persistence in soil do not suggest major problems resulting from its disposal in soil. Since it is moderately persistent and moderately mobile, care would be required to prevent its leaching into planted areas before it is degraded.

CDAA

CDAA is the common name for 2-chloro-*N,N*-di-2-propenylacetamide, a selective pre- and post-emergence herbicide introduced by Monsanto Co. in 1957 as Randox. An oily amber liquid with a boiling point of 74°C at 0.3 mm mercury, CDAA decomposes at 125°C and has a vapor pressure of 9.4×10^{-3} mm mercury at 20°C. Its water solubility is 20,000 ppm at 25°C and 200,000 ppm in kerosene at 49°C. It is synthesized by reacting chloracetyl chloride with diallylamine.

Degradation:

Pseudomonas striata degraded 36.8 percent, and an *Achromobacter* species degraded 24.5 percent, of CDAA during six days' incubation with soil (Kaufman and Blake 1973). Dairy cows which were fed five ppm of CDAA for four days excreted no intact CDAA in their milk, feces or urine (St. John and Lisk 1974). Microbial degradation is thought to be the major detoxifying process in soil, with chemical hydrolysis secondary (WSSA 1974).

Transport:

Gentz (1960) considered CDAA to leach little, and noted that soil-adsorbed CDAA retains its phytotoxicity. Deming (1963) noted that CDAA leaches through soil, but did not present data. The adsorption of CDAA to soil is directly proportional to soil colloid content and, in the absence of adsorption, four lbs/A (4.48 kg/ha) of CDAA volatilized in less than eight hours at temperatures above 7°C (WSSA 1974). Volatilization increased with increasing water saturation of soil colloids and with decreasing organic matter content of soil; on occasion, volatilization decreased with increasing temperature (Deming 1973). No data were available on transport between soil and water.

Persistence:

CDAA applied to soil at eight lbs/A (8.96 kg/ha) persisted four weeks in moist soil and somewhat longer in dry soil. Persistence was greater in soil at 80°F or 100°F than at 40°F or 60°F (Gantz 1960). Increasing temperatures decreased its persistence in moist soil (Otten *et al.* 1957).

Within three months, 160 ppm CDAA in soil had lost all phytotoxicity (Sheets 1959). The usual length of persistence of agricultural levels of CDAA is considered to be less than five weeks (Sweet *et al.* 1958) or three to six weeks (WSSA 1974).

Effects on Non-Target Species:

In the laboratory, unspecified levels of CDAA inhibited nitrification in soil (Otten *et al.* 1957b). In culture, *Rhizoctonia*, *Sclerotium rolfsii*, and *Sclerotium bataticola* were completely inhibited by 24 hours' exposure to two lbs/A (2.24 kg/ha) of CDAA, suggesting it is fungicidal at agricultural levels (Bain 1961). *Streptomyces* was not affected by CDAA at levels of six to 300 lbs/A (6.72-336 kg/ha) (Pimentel 1971).

The LC_{50} for quail was reportedly greater than 5620 ppm, and for rainbow trout and bluegills, the median tolerance levels were 2.0 ppm and 6.6 ppm, but no length of exposure was given (WSSA 1974). The acute oral LD_{50} of CDAA to rats was 750 mg/kg (WSSA 1974).

Conclusions:

The short persistence of CDAA would make it suitable for soil disposal but for the complete lack of data concerning its degradation products, their persistence and their effects on soil processes. Data on transport through soil are also insufficient for valid conclusions to be drawn.

Chloramben

Chloramben is the common name for 3-amino-2,5-dichlorobenzoic acid, a selective pre-emergence herbicide introduced in 1958 by Amchem Products, Inc. as Amiben or Vegiben. Chloramben is a white, odorless amorphous powder which melts at 201°C and has a water solubility of 700 ppm at 25°C. It is synthesized by nitration of the 2,4-dichlorobenzoic acid to produce 2,4-dichloro-3-nitrobenzoic acid, which is reduced to produce chloramben.

Degradation:

Chloramben is degraded by microorganisms, but no specific species or products were identified (WSSA 1974). Detoxification in highly organic soils is slow and not affected by pH within the normal soil range (Corbin and Upchurch 1967). In aqueous solution chloramben is decomposed by sunlight or ultraviolet light: when a 10^{-3}M aqueous solution (206 ppm) was exposed to sunlight, 49 percent remained after six hours. Exposed to a sun lamp, 51 percent persisted for six hours. In glass jars, 4 lbs/40 gal. (1,200 ppm) water was not significantly decomposed by exposure to sunlight for six hours, and the triethylamine salt was less sensitive to sunlight than pure chloramben (Sheets 1963). No data on physical or chemical decomposition were available.

Transport:

Chloramben was less readily adsorbed by activated carbon than were CIPC, trifluralin, 2,4-D or diphenamid (Coffey and Warren 1969). It is little adsorbed by soils in comparison with most herbicides, but adsorbs more readily to organic soils than to sand or clay (Donaldson and Foy 1965). In consequence of its slight adsorption, chloramben leaches readily (Donaldson and Foy 1965) even in muck albeit more in sand or silt loam (Eshel and Warren 1967). On a scale of six, chloramben was ranked 5.2

in silty clay loam and 5.7 in sandy loam, both indicating high mobility. Only dicamba was more mobile on these soils (Harris 1967).

Persistence:

In silty clay loam at temperatures between 15° and 35°C, chloramben was less persistent at higher temperatures. Although the usual persistence of chloramben in soil is six to eight weeks (WSSA 1974) and the usual period of phytotoxicity in Nebraska soils is four to six weeks, no degradation of chloramben was seen after 11 months in moist soil at 15° C. At 25°C and 35°C, 42 and 46 percent decomposed in eleven months, including a lag period of five to six months (Burnside 1965).

Effect on Non-Target Species:

At agricultural levels, chloramben did not affect soil respiration (Corke and Robinson 1960), mobile nitrates, phosphates, or potassium compounds, soil microorganisms or the redox enzymatic activity of soil (Geshtovt *et al*. 1974). At 0.4 kg/ha Pashkin (1971) observed an increase in soil nitrates and soil potassium, and four to six kg/ha depressed soil catalase for ten days (Soldatov *et al*. 1971). At five kg/ha, *Azotobacter* growth was inhibited one month after application of chloramben (Shkola 1970). Of crayfish exposed to 0.3 to 3.0 mg/liter (0.3-3.0 ppm) for an unspecified length of time, one third survived (Sestokas 1967), while up to 1000 ppm of chloramben in 60 percent sucrose was not toxic to bees (Morton and Moffett 1972). The acute oral LD_{50} of chloramben to rats was 3500 mg/kg (WSSA 1974) or 5620 mg/kg (Firantal 1971).

Conclusions:

There is not enough information on the degradation of chloramben, or on its persistence in soil, to permit conclusions as to the feasibility of its disposal in soil.

2,4-D and 2,4,5-T

2,4-D is the common name for the systemic herbicide 2,4-dichlorophenoxyacetic acid, introduced by Amchem Products, Inc. in 1942. Salts and esters of 2,4-D are its most common herbicidal formulations. The free acid is white, crystalline, and odorless if pure; it has a melting point of 140° to 141°C for pure 2,4-D, 135° to 138°C for technical 2,4-D, and a boiling point of 160°C at 0.4 mm mercury. Its solubility at 25°C is 900 ppm in water, 850,000 ppm in acetone and 1,300,000 ppm in ethanol. It is produced by the interaction of 2,4-dichlorophenol and sodium monochloracetate. The sodium salt of 2,4-D has a water solubility of 25,000 ppm at room temperature and is soluble in aqueous alkali and in alcohol, but insoluble in petroleum oils. Martin (1968) lists the many other salts of 2,4-D with their melting points and water solubility. The most common ester used is the isopropyl ester, an almost colorless liquid with a boiling point of 130°C at one mm mercury.

2,4,5-T is the common name for the herbicide 2,4,5-trichlorophenoxyacetic acid, introduced by Amchem Products, Inc. in 1944. The acid is a white, crystalline solid with a melting point of 154° to 155°C, a decomposition temperature above 200°C and a water solubility of 238 ppm at 30°C. Its solubility in ethanol is 590 ppm at 50°C (WSSA 1974). The salts of 2,4,5-T are soluble in petroleum oils. The technical acid is 98 percent pure with a melting point of 150° to 151°C (Martin 1968). 2,4,5-T is produced by the interaction of sodium monochloracetate with 2,4,5-trichlorophenol.

Degradation:

Biological--Two modes of degradation have been established for 2,4-D: dechlorination (Rogoff and Reid 1956, Bell 1957, 1960) and breakdown of the side chain (Taylor and Wain 1962, Steenson and Walker 1957). Warm, moist conditions and the addition of organic matter to the soil accelerate 2,4-D disappearance (Loos 1969). Audus (1949, 1950, 1951, 1952) conclusively established the microbial degradation of phenoxyacetic acid herbicides. Data on the degradation of 2,4-D and 2,4,5-T by soil microorganisms are shown in Table 3. Bell (1957) observed a lag before *Achromobacter* degraded 2,4-D, and suggested that the necessary enzymes were induced. Altom and Stritzke (1973) reported a five day lag before 2,4,5-T degradation began in forest soil, and no lag, or a less than five day lag, for 2,4-D degradation. While 2,4-D was equally rapidly degraded in all three soils, 2,4,5-T was more rapidly degraded in soil taken from under grass than from under trees.

Rogoff and Reid (1954) isolated a *Corynebacterium* which dechlorinated 1,000 ppm of the sodium salt of 2,4-D in as little as three days, and 80 percent of 1,000 ppm in solution at a pH of 7.1, within four hours. A *Flavobacterium* isolated by MacRae and Alexander (1963) cleaved the side chains of 2,4-dichlorophenoxypropionic acid, but not of 2,4-D; longer side chains were more readily degraded than the propionic moiety, and odd-numbered carbon-chains were more readily degraded than even-numbered chains. A similar result was obtained by Taylor and Wain (1962), who concluded that in the *ortho/para* position, Cl did not interfere in side chain cleavage, while a Cl at the *para* position inhibited *ortho*-Cl removal. In their study, *Nocardia coeliaca*, *Pseudomonas*, and *Micrococcus* successfully degraded *omega*-phenoxyalkane carboxylic acids while *Achromobacter* and *Flavobacterium* did not.

Faulkner and Woodcock (1965) reported that bacterial degradation of phenoxyacetic acids included ring fission while fungal detoxification consisted of nonspecific hydroxylation of the ring without fission. An increase in the speed of degradation by microorganisms occurred with repeated 2,4-D applications (Torstensson et al. 1975). Skryabin et al. (1974), however, were unable to culture microorganisms using 2,4-D as the sole source of carbon and concluded that 2,4-D was cometabolized to the extent of partial side chain oxidation, but that the aromatic

Table 3. DEGRADATION OF 2,4-D BY SOIL MICROORGANISMS

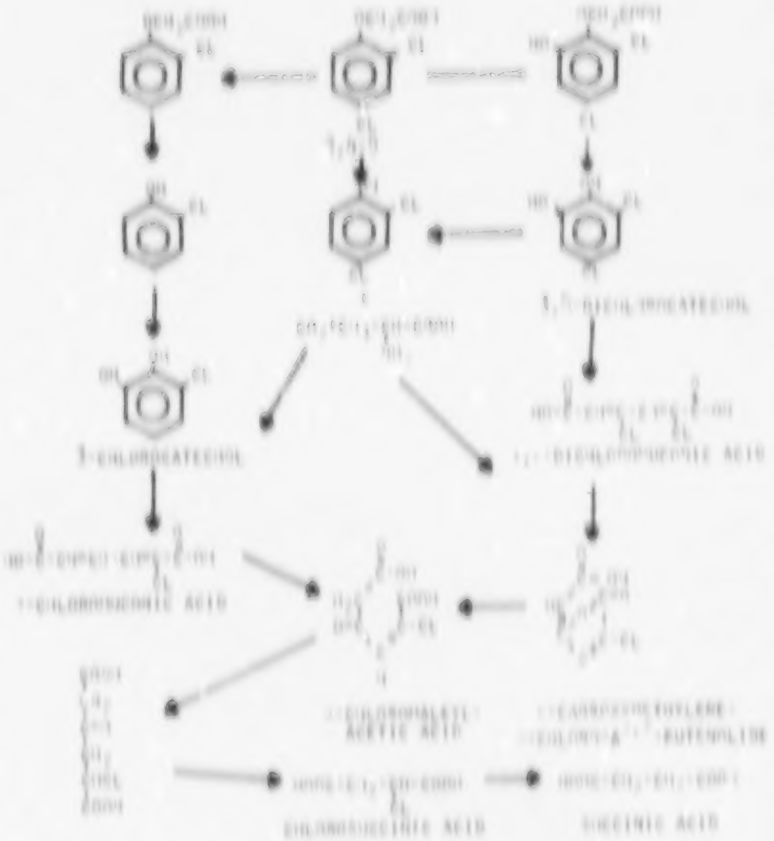
Organism	Products Identified	Reference
2,4-D		
Corynebacterium	chloride ion	Rogoff and Reid 1956
Flavobacterium peregrinum	2,4-dichlorophenol 4-chlorocatechol	Steenson and Walker 1957
Achromobacter	2,4-dichlorophenol 4-chlorocatechol	Ibid.
Arthrobacter	chloride ion	Bell 1957, 1960
Flavobacterium	none	Burger et al. 1962
Aspergillus niger	2,4-dichloro-5-hydroxyphenoxyacetic acid 2,5-dichloro-4-hydroxyphenoxyacetic acid	Faulkner and Woodcock 1964, 1965
Aspergillus niger	Dechlorinated phenol	Arnold et al. 1966
[illegible]	2-(2,4-dichlorophenoxy)ethanol	Nakajima et al. 1973
[illegible]	2-(2,4-dichlorophenoxy)ethanol	Ibid.
[illegible]	2-(2,4-dichlorophenoxy)ethanol	Ibid.
2,4,5-T		
Brevibacterium sp.	inorganic chloride 3,5-dichlorocatechol?	Horvath 1971

nucleus was intractable. Cultures of planktonic algae removed significant amounts of 2,4-D from cultures but its fate was not ascertained (Butler et al. 1975).

Phenoxyalkanoic acids with a Cl in the *meta* position, such as 2,4,5-T, are considerably more resistant to metabolism (Alexander and Aleem 1961, Jensen 1963). When 30 ug/ml (30 ppm) of 2,4-D and 2,4,5-T were placed in nutrient solution, 2,4-D was degraded within ten days, while 2,4,5-T persisted unaltered for three months (Whiteside and Alexander 1960). The pathways of microbial metabolism of 2,4-D are shown in Figure 2. When five tons per acre (11,227 kg/ha) of 2,4-D and 2,4,5-T were mixed with soil, CO_2 evolution was depressed by the pure chemicals, but enhanced by formulated compounds, suggesting that the carriers are readily biodegradable (Stojanovic et al. 1972a). The degradation of the 2,4,5-T contaminant 2,3,5,7-tetrachloro-dibenzo-*p*-dioxin (TCDD) in soil has not been demonstrated (Helling et al. 1973).

The fate of 2,4-D and 2,4,5-T in animals has been reviewed by Paulson (1975) who concluded that the nature of the metabolites in tissues, urine, and feces is not well understood. Neither 2,4-D (Bache et al. 1964) nor 2,4,5-T (St. John et al. 1964) is excreted in cows' milk. In dogs, a 5 mg/kg dose of 2,4,5-T had a plasma half-life of 77 hours and a body-clearance half-life of 86.6 hours. In rats the plasma clearance half-life ranged from 4.7 hours for a 5 mg/kg dose to 25.2 hours for 200 mg/kg. The body clearance half-life ranged from 13.6 hours for 5 mg/kg to 28.9 hours for 200 mg/kg (Piper et al. 1973). The half-life of an unspecified level of radioactive 2,4,5-T was 57 hours in newborn rats but 3.4 hours in adult female rats (Fang et al. 1973). Five men excreted unaltered 2,4,5-T in their urine with a 23.1 hour plasma clearance half-life and a 23.06 hour body clearance half-life (Gehring et al. 1973).

Photolytic--In the presence of UV light and of photo-activated riboflavin, 2,4-D was converted to 2,4-dichlorophenol and other phenols. The


3,5-DICHLOROCATECHOL
3-CHLOROCATECHOL
2-CHLOROMUCONIC ACID
2-CHLOROMALEYLACETIC ACID
CHLOROSUCCINIC ACID
SUCCINIC ACID

PROPOSED PATHWAYS OF MICROBIAL METABOLISM OF 2,4-D
(AFTER MENZIE 1974)

Figure 3

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precise composition of the degradation products depended on the initial concentration and pH of the solutions (Bell 1956). Ultraviolet light above 290 nm converted the water-dispersed ethyl and 2-methylheptyl esters of 2,4-D to 2,4-D acid and hydrogen chloride. Dechlorination was light-dependent, while the side chain cleavage was essentially a dark reaction catalyzed by the HCl produced by dechlorination. The yields were sufficiently great to suggest that these reactions are significant under field conditions (Binkley and Oakes 1974). Baur and Bovey (1974) noted that 2,4-D in so-called polymer formulations was more susceptible to degradation by long-wave UV light than were conventional 2,4-D formulations. Photolytic degradation of 2,4-D on silica gel chromatographic plates was enhanced slightly but definitely by xanthone, benzophenone, and 4,4'-dichlorobenzophenone, and more extensively by anthraquinone (Ivie and Casida 1971).

Long-wave (356 nm) UV light decomposed the potassium salt of 2,4,5-T, but not 2,4,5-T acid, at 30° and 60°C (Baur *et al.* 1973). In water, 2,4,5-T was very slowly photolyzed to 2,4,5-trichlorophenol and 2-OH-4,5-dichlorophenoxyacetic acid. Some 4,6-dichlororesorcinol, 4-chlororesorcinol, and 2,4-dichlorophenol were also formed, as well as 4-chlororesorcinol and unidentified polymeric compound. No 2,3,6,8-tetrachlorodibenzo-p-dioxin (TCDD) was detected (Crosby and Wong 1973). TCDD was photolytically extremely stable in soil, and almost as stable in water (Helling *et al.* 1973).

Chemical and physical--In natural surface waters in the laboratory, the isopropyl, isooctyl and isobutyl esters of 2,4-D were hydrolyzed to the free acid and corresponding alcohol in nine days. The alcohols decomposed further, but 2,4-D acid persisted for up to 120 days. After considerable microbial adaptation, lake muds decomposed 81 to 88 percent of 2,4-D within 24 hours (Aly and Faust 1964).

Complete degradation of 2,4-D and 2,4,5-T occurred in the presence of liquid ammonia plus either metallic sodium or lithium (Kennedy *et al.*

1972a). Partial decomposition of 2,4-D was achieved with 70 percent $Ca(ClO)_2$, 8*N* NaOH and 18*N* H_2SO_4. Partial decomposition of 2,4,5-T occurred in the presence of 8*N* NaOH and 18*N* H_2SO_4 (Kennedy et al. 1972b). Storage of 2,4-D in cardboard boxes resulted in a ten percent loss after three years, and 30 percent after six years but glass containers decreased these losses (Chorbadzhiiska 1966).

Polymeric formulations were less easily destroyed by heat than were conventional formulations of 2,4-D (Baur and Bovey 1974). Hee and Sutherland (1974) determined that the methylamine and *n*-butylamine salts of 2,4-D decomposed to form maximum amounts of the amide at 140° to 150°C, while the dimethylamine salt required temperatures above 160°C for amide formation and the *n*-dodecylamine and *n*-tetradecylamine salt formed amides most rapidly between 150° and 170°C. Other decomposition products included 2,4-dichlorophenol, imines and lactones. The amides of 2,4-D decomposed above 200°C.

When 2,4-D and 2,4,5-T were heated in muffle furnaces, over 99 percent of both compounds had decomposed at temperatures below 600°C. For 2,4,5-T, 37.4 percent decomposed below 200°C (Kennedy et al. 1969). Heating 2,4-D or 2,4,5-T to 900°C resulted in formation of the volatile compounds carbon monoxide, carbon dioxide, chlorine, hydrogen chloride gas and oxygen (Kennedy et al. 1972a, 1972b). Although the thermal degradation of both 2,4-D and 2,4,5-T is eminently feasible, the possibility of dioxin production during pyrolysis makes it inadvisable without rigorous monitoring of the conditions.

Transport:

Of ten herbicides, 2,4-D was least readily soil adsorbed. The order was: 2,4-D ≤ fluometron < prometone < simazine, atrazine and diphenamid < prometryne << chlorpropham << trifluralin << benefin (Scott and Phillips 1972). On clays, the sodium salt of 2,4-D was least readily adsorbed on bentonite and most readily on kaolin. Both the butyl, *iso*-propyl, and *iso*octyl esters were least readily adsorbed on kaolin and most readily on bentonite. Illite was intermediate in all cases (Faust and Aly 1963). The level of adsorption of 2,4-D on clay is always low (Weber *et al.* 1965, Scott and Lutz 1971, Grover 1973) but increased to some extent as pH increased (Helling 1971b, Frissel and Bolt 1962). Salting out occurred at high concentrations of 2,4,5-T or 2,4-D (Frissel and Bolt 1962). On goethite, which is positively charged, 2,4-D was maximally adsorbed at low ionic strength near its pK of 2.73 but its ready desorption could lead to rapid leaching (Watson *et al.* 1973). Adsorption on montmorillonite is physical (Khan 1974a, 1974c).

On humic acid, 2,4-D is probably also adsorbed physically rather than chemically (Khan 1973a). Esters of 2,4-D were more rapidly hydrolyzed to the free acid in soil than in aqueous solution, and effectively behaved like the acid in moist soil (Grover 1973). Organic matter is the major adsorbent of 2,4,5-T in soil (O'Connor and Anderson 1974). Both 2,4-D and 2,4,5-T are minimally adsorbed by near neutral prairie soils and strongly adsorbed by peat (Grover and Smith 1974).

As expected from this low adsorptiveness, 2,4-D is characterized as a mobile herbicide (Helling 1971a). Nevertheless, little 2,4-D leached more than five centimeters during rice field irrigation (Sokolov *et al.* 1974). In North Carolina soil, 90 percent of 2.24 kg/ha of 2,4,5-T had disappeared from the soil without penetrating significantly below 7.5 cm and without moving more than 12 meters downhill. The low rate of downhill movement was correlated with a low level of runoff (Lutz *et al.* 1973). In calcareous silty loam in greenhouses, ten cm of irrigation resulted in 35 cm leaching by 2,4,5-T (O'Connor and Wierenga 1973). When plots treated with 2.2 or 4.4 kg/ha of 2,4-D were watered with simulated rains of varying intensity, 26 percent of the 2,4-D butyl ester, but only five percent of the amine salt, were washed off by the strongest simulated rain, calculated as occurring once in a century. For a storm calculated to occur once a year, 13 percent of the butyl ester and four percent of the amine salt were washed off. Most of the

2,4-D remained in the top 7.5 cm of soil. TCDD, the dioxin contaminant of 2,4,5-T, is highly immobile in soil (Helling et al. 1973).

Between soil and water--In a survey of western streams, 2,4,5-T was the most frequently encountered contaminant, while 2,4-D reached the highest levels: 0.99 ug/liter (0.99 ppb) (Schulze et al. 1973). Treatment of a pond with 2,4-D resulted in water residues of up to 0.067 ppm and persisting 24 days, but soil residues persisted for over 85 days, decreasing from 4.96 ppm on the first day to 0.1 ppm on the 95th (Frank and Comes 1967). The washoff from cultivated fallow sandy loam soil contained no more than one ppm of the amine salt of 2,4-D but up to 4.2 ppm of the isooctyl and butyl esters (Barnett et al. 1967). Leaching is expected to be great if rain is heavy and occurs soon after 2,4-D is applied (Scott and Lutz 1971). On grass-covered Houston black clay, five sprayings of 2,4,5-T and picloram at 1.12 kg/ha resulted in soil levels of up to 238 ppb, while runoff water contained 400 to 800 ppb of pesticides if heavy rain followed application of the herbicides. If rain was delayed for over a month, runoff contained less than five ppb of the herbicides (Bovey et al. 1974). After two applications per year for three years, 2.24 kg/ha of a 1:1 mixture of picloram and 2,4,5-T had contaminated groundwater. Even a single treatment of 1.12 kg/ha of the mixture led to water levels of one to four ppb picloram (Bovey et al. 1975).

When 2,4-D was tested in the model ecosystem, no 2,4-D residues were found in any of the organisms after 33 days (Sanborn 1974, Metcalf and Sanborn 1975).

Persistence:

The average losses per day of 2,4-D and 2,4,5-T are shown in Table 4. In all studies in which both herbicides were examined 2,4,5-T was more persistent, which Alexander and Aleem (1961) attributed to meta-Cl. O'Connor and Wiurenga (1973) noted that repeated application enhanced

Table 4. PERSISTENCE OF 2,4-D, 2,4,5-T AND TCDD IN SOIL AND WATER

% Residue	# Days	Mean loss day (%)	Conditions	Reference
2,4-D				
0	>870	<0.11	90 cm depth in soil	Lavy et al. 1973
0	>510	<0.20	40 cm depth in soil	Ibid.
0	330	0.30	butyl, butoxyethyl, chlorocrotyl esters	Ashkharova 1973
0	>150	<0.67	15 cm depth in soil	Lavy et al. 1973
0	>120	0.83	water	Faust and Aly 1967
0	50	2.00		Kostrowska et al. 1972
0	35	2.86	bioassay	Sweet et al. 1958
50	4	12.50	diethanolamine salt	Altom and Stritzke 1973
2,4,5-T				
0	>120	<0.83	soil	Alexander and Aleem 1961
0	85	1.18	80 ppm	O'Connor and Wierenga 1973
50	20	2.50	3 soils; ethanolamine salt	Altom and Stritzke 1973
TCDD				
50	365	0.14		Helling et al. 1973

the degradation of 2,4,5-T while high levels (64 kg/ha) enhanced persistence. The maximum rate of degradation resulted in decomposition of 2,4,5-T at 40 ppm in 43 days in calcareous silty loam. Altom and Stritzke (1973) noted a lag of more than five days before 2,4,5-T degradation began but no lag for 2,4-D degradation.

When 2,4-D was placed in field pits, the rate of degradation decreased with increasing soil depth. In anaerobic soils, degradation was extremely slow at all depths (Lavy et al. 1973). The optimum pH for 2,4-D detoxification was 5.3 (Corbin and Upchurch 1967).

The dioxin contaminant of 2,4,5-T, TCDD, persisted less than six years in soil when 1,060 kg/ha of 2,4,5-T were applied to Lakeland sand (Helling et al. 1973). The propyleneglycolbutylether ester of 2,4-D remained in containers at the rate of 24 ml per five gallon drum and 166 ml per 55 gallon drum and was more persistent than emulsifiable concentrates of parathion (Archer 1975).

Effects on Non-Target Species

Microorganisms--The effects of 2,4-D on soil processes and soil microorganisms are shown in Tables 3 through 11. Among the consequences of 2,4-D application are an increase in the organic carbon content of soil (Oh 1973) and an irreversible genetic effect (*sic*) on *Streptomyces globisporus* and *Streptomyces fasciculus* (Kiss 1966). Resistance to 2,4-D can be selected for (Sokolov et al. 1975). *Aerobacter aerogenes* growth was depressed for 25 generations in the presence of 1,000 ppm of 2,4-D with a concomitant increase in the length of the lag phase before 2,4-D was degraded. At 5,000 ppm, however, the lag phase decreased even though growth was inhibited for 91 serial subcultures (Dean and Law 1964). Strains of *Azobacter* which were selected for resistance to 2,4-D sometimes lost their ability to produce vitamins B_1 and B_2 (Hammouda 1974). Facultatively anaerobic bacteria were more sensitive to 2,4-D under anaerobic than under aerobic conditions, while gram negative

Table 5. EFFECTS OF 2,4-D ON SOIL PROCESSES

Parameter	Effect	Level Original	Level kg/ha	Time obs. (days)	Conditions	Reference
Respiration	0	ag levels (<2 lbs/A)	<2.24		Warburg apparatus	Kalinska 1967a
	0	100 x ag levels	<224		clay loam, sandy clay loam	Ruffin 1974
	–	2,20 kg/ha	2,20		sandy podzolic soil	Eliev 1973
	–	2 lbs/A	2.24	32	silty clay loam	Teater et al. 1958
	–	8 lbs/A	8.96	32	silty clay loam	Ibid.
	–	32 lbs/A	35.84	32	silty clay loam	Ibid.
	0	ag levels	<2.24		silty clay loam	McCalla et al. 1962
	0	10;100 x ag levels	22.4;224		silty clay loam; lab	Ibid.
Ammonification	+	1 lb/A	1.12		field, post-emergence	Singh 1971
	+	1.1 kg/ha	1.1	<84	amine ester; clay loam	Castro 1964
	+	>0.25%	2125		black or sandy loam	Jones 1956

+ = stimulation
0 = no effect
– = inhibition

Table 5. Cont'd

Parameter	Effect	Level Original	Level Kg/ha	Time obs. (days)	Conditions	Reference
Nitrification	0	1 lb/A	1.12		field; pre- or post-emergence	Singh 1971
	+	3 kg/ha	3.00		soils	Dukhanin and Kolosova 1959
	+			75	sandy or chernozem soil	Dukhanin and Kolosova 1962
	+					Pashkin 1971
	-	8.32 lbs/A	9.32	30		
	-	ag levels	<2.24		silty clay loam; lab	McCalla et al. 1962
	+	10 x ag levels	<22.4		" " " "	Ibid.
	+	100 x ag levels	224		" " " "	Ibid.
	0	2.1 kg/ha	2.1		clay loam, amine ester	Castro 1964
	+				clay loam; isopropyl ester	Vicario 1972
	-				leached chernozem	Svyatskaya 1972
	+	<0.25%	5600		black or sandy loam	Jones 1956
Available phosphate	+	3 kg/ha	3.00		soils	Dukhanin and Kolosova 1959
	+			75	sandy or chernozem soils	Dukhanin and Kolosova 1962
	0	2.1 kg/ha	2.1		clay loam, amine ester	Castro 1964
	+				clay loam, isopropyl ester	Vicario 1972
Soil organic content	+					Oh 1973

+ = stimulation   0 = no effect   - = inhibition

Table 6. EFFECT OF 2,4-D ON SOIL ENZYMES AND SOIL ELEMENTS

Parameter	Effect	Level Original	Level kg/ha	Time observed	Conditions	Reference
Enzyme levels						
Urease	+	1 kg/ha	1	grow. season	podzolic loam	Zinchenko et al. 1969
	+			5 days		Zinchenko and Osinskaya 1969
	–					Svyatskaya 1972
Catalase	–	1 kg/ha	1		podzolic loam	Zinchenko et al. 1969
	–			40 days		Zinchenko and Osinskaya 1969
Saccharase	–					Svyatskaya 1972
S-oxidation	+	>0.25%	5600		soil	Jones 1956
Potassium	+					Pashkin 1971
	0	2.2 kg/ha	2.2	<12 weeks	amine ester, clay loam	Castro 1964
Phosphorus	+				isopropyl ester	Vicario 1972
	0	2.2 kg/ha	2.2	<12 weeks	amine ester, clay loam	Castro 1964
Calcium	+				isopropyl ester	Vicario 1972
	0			<12 weeks	amine ester, clay loam	Castro 1964
Magnesium	0	2.2 kg/ha	2.2	<12 weeks	" " " "	Ibid.
Manganese	0	2.2 kg/ha	2.2	<12 weeks	" " " "	Ibid.
Iron	0	2.2 kg/ha	2.2	<12 weeks	" " " "	Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 7. EFFECTS OF 2,4-D ON CLASSES OF BACTERIA

Organism	Effect	Level Original	Level PPM	Time obs. (days)	Conditions	Reference
Bacteria: all	0	2 kg/ha	0.9		dark chestnut soil	Ilyaletdinov and Zharnikova 1963
	0	ag levels	<2			Jensen 1963
	-					Venkataraman and Rajyalakshmi 1971
	0	1 lb/A	0.5		field, pre-, post-emergence	Singh 1971
	-	300-600 ppm	300-600		" " "	Ibid.
	0	.004-0.22	40-1000	>25		Scheffer et al. 1952
Cellulolytic	0	2 kg/ha	0.9		dark chestnut soil	Ilyaletdinov and Zharnikova 1963
	+			>300		Gerlinovich et al. 1962
	+	1.7 mg/kg	1.7			Abueva and Bagaev 1975
Fluorescent	-			>300	peat bog soil	Gerlinovich et al. 1962
Nitrifying	+	1 kg/ha	0.45		acid, enters in rhizosphere	Kozlova and Ilikaeva 1963
	-				36 strains in nodules	Vinitskova et al. 1965
	+	1.7 mg/kg	1.7		soddy-podzol	Abueva and Bagaev 1975
Denitrifying	-	6 kg/ha	1.7		soddy-podzol, NH_4 salt	Ibid.
Ammonifying	-	1 kg/ha	0.45		rhizosphere	Kozlova and Ilikaeva 1963
	+	1.7 mg/kg	1.7		soddy-podzolic soil	Abueva and Bagaev 1975

+ = stimulation   0 = no effect   - = inhibition

Table 8. EFFECTS OF 2,4-D ON CLASSES OF FUNGI

Organism	Effect	Level: Original	Level: PPM	Time obs. (days)	Conditions	Reference
Fungi: all	0	2 kg/ha	0.9		dark chestnut soil	Ilyaletdinov and Zhurakova 1963
	+			>300	peat bog soil	Gartsevich et al. 1962
	0	4 kg/ha	2.7		sodic-podzol, NH_4-salt	Aburova and Bagaev 1975
	+					Bakalivanov 1972
	-	300,600 ppm	300,600			Singh 1971
	+	1 lb/A	0.5			Ibid.
	+	$\leq$0.304%	$\leq$40	$\leq$35	soils	Schaffer et al. 1952
	-	$\geq$0.1%	$\geq$1000	$\leq$35	soils	Ibid.
	+	100 ppm			culture	Roberts and Bollen 1955
	-	10,000 ppm	100-10,000		culture	Ibid.
Actinomycetes	-					Venkataraman and Rajyalakshmi 1971
	+	$\leq$0.304%	$\leq$40		soils	Schaffer et al. 1952
	-	$\geq$0.1%	$\geq$1000		soils	Ibid.

+ = stimulation 0 = no effect - = inhibition

Table 9. EFFECTS OF 2,4-D ON SPECIES OF SOIL MICROORGANISMS: BACTERIA

Organism	Effect	Level Original	 PPM	Conditions	Reference
Azotobacter	-			63 strains	Hammouda 1974
	0	1 lb/A	0.5		Singh 1971
	-	300, 400 ppm	300, 400		Ibid.
	0	2 kg/ha	0.9	dark chestnut soils	Ilyaletdinov and Zhgurkova 1963
A. chroococcum	-	0.1-1000 ppm	≤1000		Cenci and Cavazzini 1973
Clostridium	-	<10 μ mole/liter	<22.1	4 species	Hart and Larsen 1966
C. pasteurianum	+	1 kg/ha	0.45		Kozlova and Dikareva 1963
	-	0.1-1000 ppm	0.1-1000		Cenci and Cavazzini 1973
Shigella sonnei	-	≥.05 mg/ml	≥50		Prasinel et al. 1962
S. ambigua	-	≥.05 " "	≥50		Ibid.
S. dysenteriae	-	≥.02 " "	≥20		Ibid.
Aerobacter aerogenes	-	<1000 mg/liter	<1000		Dean and Law 1964
Bacillus subtilis	-	≤1 μM/ml	≤0.22		Hart and Larsen 1966
	0	1 mg/ml	1000		Thomas et al. 1973

+ = stimulation
0 = no effect
- = inhibition

Table 9. Cont'd

Organism	Effect	Level Original	 PPM	Conditions	Reference
B. megaterium	-	1 µM/ml	≤0.21		Hart and Larson 1966
Thiobacillus thioparus	-	0.1-1000 ppm	0.1-1000		Cenci and Cavazzini 1973
Ferrobacillus ferrooxidans	+	1-1000 ppm	0.1-1000		Ibid.
Desulfovibrio desulfuricans	-	0.1-1000 ppm	0.1-1000		Ibid.
Methanococcus vanielii	-	0.1-1000 ppm	0.1-1000		Ibid.
Corynebacterium fascians	-	1 µM/ml	≤0.21		Hart and Larson 1966
Arthrobacter pascens	-	1 µM/ml	≤0.21		Ibid.

+ = stimulation
0 = no effect
- = inhibition

Table 10. EFFECTS OF 2,4-D ON SPECIES OF SOIL MICROORGANISMS: FUNGI

Organism	Effect	Level Original	 PPM	Time observed	Conditions	Reference
Penicillium sp.	–	1000 ppm	1000		on agar	Pommer 1964
Trichoderma viride	–	1000 ppm	1000		on agar	Ibid.
Trichoderma	–			>10 mos.		[illegible] *et al.* 1962
Rhizopus nigricans	–	1000 ppm	1000		on agar	Pommer 1964
Fusarium oxysporum	–	.12, .25 mg/ml	120,250			Szegi 1973
Aspergillus niger	–	10, 50 ppm	10,50			Arnold *et al.* 1966
A. oryzae	+	1-100	1-100			Cenci and Cavazzini 1973
Saccharomyces cerevisiae	–	0.1-100 ppm	0.1-100			Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 11. EFFECTS OF 2,4-D ON SPECIES OF SOIL MICROORGANISMS: ALGAE

Organism	Effect	Level Original	 PPM	Time observed	Conditions	Reference
Chlorella vulgaris	–	0.1-100 ppm	0.1-100			Ibid.
C. pyrenoidosa	0	1 mg/ml	1000			Thomas et al. 1973
Nostoc sp.	–	>100 ppm	100			Venkataraman and Rajyalakshmi 1972
N. muscorum	–	0.1-100 ppm	0.1-100		chlorophyll production	Cenci and Cavazzini 1973
Scenedesmus quadricauda	–	0.1,1.0 mg/ml	100,1000		static culture	Stadnyk et al. 1971
Anabaena sp.	–	>100 ppm	>100			Venkataraman and Rajyalakshmi 1972
Tolypothrix tenuis	–	>100 ppm	>100			Ibid.
Aulosira fertilissima	–	>100 ppm	>100			Ibid.
Chlorococcum	–	400 ppm	400			Arvik et al. 1971
Cylindrospermum licheniforme	–	400 ppm	400			Ibid.

+ = stimulation
0 = no effect
– = inhibition

bacteria within each metabolic group were less sensitive than gram-positive bacteria (Hart and Larson 1966). The sodium salt of 2,4-D was considered toxic to microorganisms in peat-podzolic soil at unspecified high levels (Mashtakov et al. 1962).

Metabolism in *Tetrahymena pyriformis* was inhibited by 0.5 to 0.9 mM of 2,4,5-T; cell division stopped for up to one hour and mitochondrial oxygen uptake slowed. Sensitivity decreased with time (Silberstein and Hooper 1975). In sandy or muddy soil five ppm of 2,4,5-T inhibited nitrification while ten to 15 ppm enhanced nitrification (Cervelli et al. 1974).

Mixtures of dalapon and 2,4-D inhibited soil respiration of soddy-podzolic soil at two or 20 kg/ha but enhanced respiration at six and 60 kg/ha (Bliev 1973). Dicamba counteracted the inhibiting effect of unspecified levels of 2,4-D on phosphorus-mobilizing organisms, nitrification, and on saccharase and urease activity in leached chernozem soils (Svyatskaya 1972).

Invertebrates--Beran and Neururer (1955) reported that 2,4,5-T, the sodium salt of 2,4-D, and the butyl esters of 2,4-D and 2,4,5-T were not toxic to bees, but Kirkor and Giese (1960) found 0.01 percent (100 ppm) of 2,4-D in sugar to be toxic to bees. Newly emerged honeybees were not acutely sensitive even to 1,000 ppm of 2,4-D or 2,4,5-T, but their mean lifespan was decreased by the esters of either acid (Morton et al. 1972a). Neither agricultural levels of 2,4-D butyl ester nor ten times agricultural levels affected soil microarthropods significantly after ten days or four months (Rapoport and Cangioli 1963). In the laboratory the order of toxicity of eight herbicides to beetles (*Bembidion*) was: sodium chlorate > dalapon > simazine plus amitrole > pyrazon > diquat > 2,4-D > chlorpropham (Mueller 1971). Treatment with 40 lbs/A (44.8 kg/ha) of an emulsifiable concentrate of the amine salt of 2,4-D did not affect the numbers of springtails, wire worms, or mites in grassland soil (Fox 1964).

No mortality was found among oysters (*Crassostrea virginica*) exposed to 0.1 ppm 2,4-D butoxyethanol ester for seven days or among pigtoe mussels (*Pleurobema cordatum*) exposed to 100 lbs/A (50 ppm) for 72 hours, to 1000 lbs/A (500 ppm) for 48 hours or to five ppm for seven days. The oysters accumulated 18 ppm of 2,4-D which was eliminated after seven days in clear water (Butler 1965). Secondary effects of 2,4-D and other herbicides in aquatic ecosystems have been reviewed by Hurlbert (1975).

Fish and amphibians--At agricultural levels, 2,4-D is not toxic to the larvivorous fish *Aplocheilus latipes* and *Danio platyrus* (Rhin and Seif 1973). Schlueter (1969) warned that 2,4-D and 2,4,5-T were toxic to fish if carelessly used. Differences in toxicity between 2,4-D and 2,4,5-T were not as great as differences between formulations: the 24 hour TL_m of 2,4-D isopropyl ester to bluegills (*Lepomis macrochirus*) was 0.9 ppm while the TL_m of the alkanolamine salts approached 900 ppm. For 2,4,5-T, the 24 hour TL_m was 1.4 ppm for the butoxyethanol ester, but 144 ppm for the dimethylamine salt (Davis and Hughes 1963, Hughes and Davis 1963). Davis and Hardcastle (1959) calculated the 24 hour TL_m of 2,4-D acid to be 350 to 390 ppm in bluegills (*Lepomis macrochirus*) and 350 to 375 ppm in largemouth bass (*Micropterus salmoides*). Commercial salts of 2,4-D and 2,4-D acid were not toxic to green sunfish (*Lepomis cyanellus*) at 5 x 10^{-4}M (110.5 ppm) but the butoxyethanol ester was (Sergeant et al. 1970).

The effects of 2,4-D and 2,4,5-T on fertilized fish eggs and fish fry of bluegills (*Lepomis macrochirus*), green sunfish (*L. cyanellus*), small mouth bass (*Micropterus dolomieu*) lake chub-suckers (*Erimyzon sucetta*) and stonerollers (*Campostoma anomalum*) also depended on formulations, with 2,4-D toxicity at two ppm for the propylene glycol butoxyethanol ester but only at 100 ppm for the sodium salt. For 2,4,5-T, the sodium salt was toxic at 50 ppm, and the isooctyl ester was toxic at one ppm if in liquid formulation, and at ten ppm if in granules (Hiltibran 1967).

In carp (*Cyprinus carpio*) hatching was delayed by five ppm of the sodium salt of 2,4-D while 50 ppm caused massive deaths of fry five days after hatching (Matlak 1972, Kamler 1972).

Birds--The acute oral LD_{50} of 2,4-D acid was greater than 1,000 mg/kg for mallards, was 472 mg/kg for pheasants, and 668 mg/kg for coturnix quail (Pimentel 1971). The LC_{50} for 2,4,5-T was greater than 5,000 ppm for coturnix quail and for mallards; and between 1,250 and 2,500 ppm for pheasants (Heath et al. 1972). The LC_{50} of 2,4-D acetamide and 2,4-D butoxyethanol ester were over 5,000 ppm for two-week old quail (Heath et al. 1972) while chicks were unaffected by 2,4-D or 2,4,5-T at up to 100 ppm (Anderson et al. 1962).

Oil solutions of the butyl esters of 2,4-D and 2,4,5-T were more toxic than aqueous suspensions when given orally to chicks (Stupnikov 1972). Roberts and Rogers (1957) fed 62 mg/kg 2,4,5-T per day to turkeys for 11 days without affecting their growth. Kenaga (1975) reviewed the data and concluded that at the usual levels of 2,4,5-T spraying, birds would not be affected adversely.

The effects of 2,4-D on avian reproduction are disputed. Dunachie and Fletcher (1967, 1970) observed a 50 percent decline in the hatchability of hens' eggs injected with 200 ppm 2,4-D (10 mg/egg), and a 30 percent decline in the hatchability of hens' eggs injected with 100 ppm (five mg/egg). Pheasants given 75 to 150 mg technical 2,4-D or its amine salt produced normal numbers of eggs with only two percent abnormalities (Solomon et al. 1973). Grolleau et al. (1974) sprayed the dimethylamine salt of 2,4-D over the eggs of quail (*Coturnix coturnix*), of grey partridges (*Perdix perdix*) and of red partridges (*Alectoris rufa*) at levels equal to 1.2, 2.4 and 6.0 kg/ha. No toxic effects were noted except a possible decrease in *Perdix* egg viability at six kg/ha, and few residues of 2,4-D were found in any of the eggs. A series of studies on the effects of 2,4-D, 2,4,5-T, picloram and DDT on chickens demonstrated that the pesticides penetrated the eggs between days five and 19, but hardly at all between days three and five (Somers et al. 1974a). Even treatment with 33.6 kg/ha of 2,4-D did not adversely affect eggs in comparison with controls (Somers et al. 1974c). Similarly negative results were obtained with studies on pheasants (*Phasianus colchicus*) in which 2,4-D mixed with picloram or 2,4,5-T actually increased the hatching rate (Somers et al. 1974b). These studies are unfortunately marred by the extraordinarily high incidence of malformations in control embryos: 12.5 to 23 percent in chicken studies (Somers et al. 1973) and 50 percent in the pheasant study (Somers et al. 1974b). Lutz and Lutz-Ostertag (1970) presented data showing that 2,4-D sprayed on pheasant, red partridge, and gray partridge eggs caused abnormalities and high mortality in the offspring although a lack of quantitative data on controls weakened this study.

Mammals--The considerable controversy over the effects of 2,4,5-T and/or its contaminant TCDD (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) has been reviewed repeatedly (Anonymous 1971, Whiteside 1971, Gribble 1974, Anonymous 1974a).

The acute oral LD_{50} of 2,4-D and 2,4,5-T in rats are 400 to 500 mg/kg and 300 to 800 mg/kg, respectively (Jones et al. 1968). Stupnikov (1962) listed the 100 percent lethal dose of the butyl acetate salt of 2,4-D as 900 mg/kg in mice, 2,000 mg/kg in rats, 1,000 mg/kg in rabbits and 1,500 mg/kg in guinea pigs. Buslovich (1962) noted that the acute oral toxicity of 2,4-D acid and its sodium salt were the same in mice, but the acid was more toxic to rats than the salt. Among the effects claimed for sublethal doses of 2,4-D are uncoupling of oxidative phosphorylation in rats (Graff 1972) and inhibition of the tuberculosis bacillus *in vitro* (Mahishi et al. 1965). Liver damage occurred in field mice given 200 mg/kg 2,4-D butyl ester orally. The damage was not always reversible and was less severe when pure rather than technical butyl ester was given (Skokova 1972).

Gehring et al. (1973) fed ten mg/kg/day of 2,4,5-T to five male human volunteers and found no clinical effects, but after three days continuous feeding plasma levels were calculated to reach a plateau at 17 times the daily intake.

In tissue culture, 0.4 mg/ml (400 ppm) of the diethylamine salt of 2,4-D caused chromosome aberrations in human fibroblasts (Berin et al. 1973) and three hours' exposure of cultured human lymphocytes to 0.1 uM or to 250 uM 2,4-D (22.1 to 55,250 ppm) caused chromosome aberrations. In mice, 100 to 300 mg/kg of 2,4-D increased the *in vivo* incidence of bone marrow chromosome aberrations (Pilinskaya 1974). No effect of 2,4,5-T or 2,4-D against Ehrlich ascites carcinoma was observed by Schultz and Norman (1965) and no increase in tumors was observed among Swedish railroad workers occupationally exposed to 2,4-D or 2,4,5-T (Axelson and Sundell 1974). Effimenko (1974) increased chromosomal aberrations in rats by feeding 0.01 to 10 mg/kg (0.01 to 10.0 ppm). These data are not sufficiently definitive to vitiate Rosival's (1970) conclusion that the effects of the phenoxyacetic herbicides on carcinogenesis and mutagenesis are unknown. In contrast, the data on the reproductive effects are unequivocal in that 2,4,5-T contaminated by greater than 0.1 ppm TCDD is teratogenic (Anonymous 1971, Anonymous 1974). Pure TCDD was both embryotoxic and teratogenic in mammals (Neubert et al. 1973). No embryotoxic effects were seen at 0.003 ppm in rats (Lucier et al. 1975). The butyl ester of 2,4,5-T is both embryotoxic and teratogenic in rats (Sokolik 1973, Konstantinova 1974a, 1974b). The acid itself, contaminated by less than 0.05 ppm dioxin (TCDD) was not teratogenic in rhesus monkeys (*Macaca mulatta*) (Dougherty et al. 1975). Lindquist and Ullberg (1971) established the accumulation of both 2,4-D and 2,4,5-T in murine yolk sacs and fetal tissues. They noted that 2,4-D dissipated faster than 2,4,5-T. Haus (1973), in his survey of pesticide residues in the environment, identified the *iso*octyl, *iso*butyl and *iso*propyl esters of 2,4-D as teratogenic in mice, and 2,4,5-T as teratogenic in rats and mice.

Conclusions:

The effects of 2,4-D and 2,4,5-T on soil and soil organisms are not sufficiently great to prohibit soil disposal of these herbicides. Since 2,4-D is readily degraded, the major concern in its disposal should be its herbicidal effects rather than environmental side affects. The same cannot be said for 2,4,5-T, which is both more persistent than 2,4-D and most probably contaminated, however slightly, with the extremely toxic and highly persistent TCDD. Disposing of large quantities of 2,4,5-T in or on soil, carries an unknown risk of TCDD contamination of soil, plants, and animals. The relative merits of burning 2,4,5-T, with the possibility of dioxin formation, or of burying it, with the probability of disseminating dioxins, cannot be determined on the basis of the data now available.

Dicamba

Dicamba is the common name for 3,6-dichloro-*o*-anisic acid, introduced by Velsicol Chemical Co. in 1959 as Banvel. Technical dicamba is a brown crystalline solid, and pure dicamba is a white, odorless crystalline solid with a melting point of 114 to 116°C, a solubility of 0.45 g/100g water (4500 ppm) and 92.2 g/100g ethanol (922,000 ppm), and a vapor pressure of 3.75×10^{-3} mm mercury at 100°C. It is formulated as the diethylamine salt with a water solubility of more than 72 g/100ml (720,000 ppm). Dicamba is synthesized from 1,2,4-dichlorobenzene by a three stage process in which trichlorobenzene is treated with methanol and sodium hydroxide to produce 2,5-dichlorophenol which, treated with carbon dioxide under pressure produces 2-hydroxy-3,6-dichlorobenzoic acid. The latter is reacted with dimethyl sulfate (Martin 1968).

Degradation:

Biological--The optimum pH for detoxification of dicamba on highly organic soils was 5.3 (Corbin and Upchurch 1967). Detoxification of 1.1 kg/ha was completed between May and October or October and May in unsterilized prairie soil. Essentially no degradation occurred in sterile soil during a four week period, but over 50 percent degradation occurred in two weeks in unsterilized soil (Smith 1973a) and 95 percent decomposed in five weeks in Regina heavy clay. Degradation of 3,6-dichlorosalicylic acid was 28 percent complete after five weeks in the heavy clay, and no 5-hydroxy-dicamba was found (Smith 1973b). Dicamba detoxification was unaffected by microbial adaptation to the herbicide MCPA (Kirkland and Fryer 1972) but occurred more rapidly as temperature and soil moisture levels increased (Burnside and Lavy 1966). Berezovskii and Mikhotin (1972) reported that 13 percent of dicamba was converted to 3,6-dichlorosalicyclic acid. The latter compound persisted for long periods (Berezovskii 1974).

Krumsdorf (1974) identified 3,6-dichlorosalicylic acid, *p*-aminobenzoic acid and benzoic acid as dicamba metabolites. In prairie soils, ^{14}C-dicamba was converted to $^{14}CO_2$ and 3,6-dichlorosalicylic acid, with the latter decomposing more slowly than dicamba. No other metabolites were detected, little or no metabolism occurred in sterilized soils and only four to five percent were lost when dicamba was incubated in soil in sealed flasks, presumably because of anaerobic conditions (Smith 1974).

In water, dicamba was most rapidly lost in unsterilized water in the presence of light. Neither vegetation nor turbidity affected the rate of loss, nor did the presence of sediments. Dissipation was presumed to be microbial (Scifres et al. 1973). Burnside and Lavy (1966) noted that dicamba was more biodegradable than either amiben or atrazine, and that little degradation occurred in autoclaved soil.

Photolytic--Dicamba is relatively resistant to ultraviolet light (WSSA 1974). Degradation was not enhanced by exposure to long wave (356 nm) UV light for seven days at 30° or 60°C (Baur et al. 1973). In conventional formulations dicamba was more susceptible to decomposition by UV light than were its so-called polymeric formulations. The ratio of volatilization to photolysis was 56:21 for polymeric dicamba, and 60.1: 37.9 for ordinary dicamba, with degradation accounting for 21 percent and 37.9 percent of the initial amounts, respectively (Baur and Bovey 1974).

Chemical and physical--Dicamba was partially decomposed by 18N sulfuric acid or 70 percent $Ca(ClO)_2$, but not by 8N sodium hydroxide, 100 percent monoethanolamine or 100 percent triethanolamine (Kennedy et al. 1972b). Dicamba was also 100 percent decomposed by metallic sodium or lithium in the presence of liquid ammonia (Kennedy et al. 1972a). When heated in a muffle furnace, over 98 percent of dicamba was decomposed at 600°C (Kennedy et al. 1969). The volatile products of burning dicamba at 900°C included carbon monoxide, carbon dioxide, chlorine, hydrogen chloride, oxygen and ammonia (Kennedy et al. 1972a, 1972b). Dicamba heated to $210^{\circ}C$ was changed to a clear liquid (Stojanovic et al. 1972b).

Transport:

Within soil--Dicamba adsorbed to kaolinite to a considerable extent, but only a small amount adsorbed to muck (Burnside and Lavy 1966). The adsorption of dicamba on montmorillonite was affected by the saturation ion and by the degree of saturation of the clay (Carringer et al. 1975). Adsorption on near-neutral prairie soils was minimal, and when several adsorbents were compared, the degree of adsorption was: charcoal > anion exchange resin > peat > cellulose triacetate (Grover and Smith 1974).

Dicamba was rated as highly mobile in both silty clay loam and sandy loam (Harris 1967) and its mobility increased as pH increased and also increased with increased flux (Helling 1971). In soil columns of 6.25 mm x 1.8 m, dicamba retention time was only 2.6 minutes, indicative of its possibly leaching to 1.5 meters in soil. In forest soils, dicamba was highly mobile even when the soil contained eight percent organic matter and 50 percent clay (Norris and Montgomery 1975).

Between soil and water--When dicamba was applied to the soil surface, more was lost to surface water from sod than from fallow soil (Trichell et al. 1968). When applied to a heavy-textured soil, two to seven percent of dicamba was lost to drainage water. Up to 5.5 times as much was lost from conventionally-tilled as from no-till plots, runoff in surface water was greater than runoff into drainage tiles, and more was lost if rain occurred soon after application than if rain was delayed (Schwab et al. 1973).

Norris and Montgomery (1975) considered stream contamination by dicamba unlikely because stream detritus and stream biota readily adsorbed the herbicide, resulting in rapid decreases in the water levels.

In the aquatic-terrestrial ecosystem, unchanged dicamba was the major component of the water after 33 days. There was no evidence of accumulation in aquatic food chains and very little uptake whatever by the organisms. Ten percent of the dicamba had been converted to 5-hydroxy-dicamba (Sanborn 1974, Yu et al. 1975, Metcalf and Sanborn 1975).

Volatilization:

Little dicamba was lost to volatilization from silty clay loam after eight weeks with relative humidities between zero and 100 percent, but 50 percent volatilized from planchets in 11 weeks (Burnside and Lavy 1966).

Persistence:

The persistence of dicamba in soil is summarized in Table 12. At pH 7.5, traces were found for three years, but persistence is usually estimated in weeks rather than in months. It is noteworthy, however, that 2,3-dichloro-salicylic acid is considerably more persistent than dicamba. Berezovskii (1974) noted its accumulation over a three year period. Krumzderov (1974) detected 3,6-dichlorosalicylic acid in oats plants sown two and three years after dicamba was applied to soil. Up to 35 percent of 3,6-dichlorosalicylic acid was adsorbed by sandy loam soil (Smith 1974). Dicamba decomposes faster in soil taken from under grass than in soil taken from under trees (Altom and Stritzke 1973) or from litter-covered forest soil than from cleared forest soil (Brady 1975), but was rapidly decomposed under all the preceding conditions. In soil under five inches of bracken litter, however, dicamba residues could be detected for three years (Parker and Hodgson 1966). In Nebraska soils, dicamba was less persistent than either picloram or 2,3,6-TBA (Burnside et al. 1971).

In water, dicamba dissipated as a logarithmic function of concentration with time, and dissipation was enhanced by light, especially in the presence of sediment (Scifres et al. 1973).

Effect on Non-Target Species:

Microorganisms--At 0.12 kg/ha, dicamba was not toxic to soil microorganisms from 26 to 80 days after soil treatment (Bezuglov et al. 1973).

Table 11. PERSISTENCE OF DICAMBA IN SOIL

% residue	# days	% lost/day	Conditions	Reference
>0	1095	0.09	pH 7.5, soil	Parker and Hodgson 1966
0	365	0.27	soil	Bereznowski 1974
<1	120	<0.82	fallow plots	Tritchell et al. 1968
0	120	0.83	1 ppm, sandy loam, 25°C	Burnside and Lavy 1966
>0	90	<1.11	California ag soils	Lange et al. 1968
0	60	1.67	1 ppm, sandy loam, 15°C	Burnside and Lavy 1966
>0	56	<1.79	bioassay	Brady 1975
10	35	2.57	Regina heavy clay	Smith 1973b
100	28	–	sterile soil	Smith 1973a
50	28	1.79	prairie soils	Smith 1974
21	28	2.82	Regina heavy clay	Smith 1973b
50	25	2.00	several soils	Altom and Stritzke 1973
36	21	3.05	Regina heavy clay	Smith 1973b
64	14	2.57	" " "	Ibid.
<50	14	>3.57	moist, unsterilized soil	Smith 1973a
70	7	10.00	Regina heavy clay	Smith 1973b
50	4	12.50	25°C, pH6	Parker and Hodgson 1966

In a sod-podzolic soil, dicamba applied at an unspecified level inhibited aerobic bacteria (Kosarev and Laptev 1972). Five tons per acre (5000 ppm) of dicamba incorporated with a calcareous loam slightly inhibited respiration and soil bacteria whether the analytical standard or the formulated liquid was used. Streptomycetes were markedly inhibited by pure dicamba and only slightly by formulated dicamba, and fungi were stimulated by pure dicamba (Stojanovic et al. 1972a).

Invertebrates--Honeybees were extremely sensitive to dicamba, with an estimated LD_{50} of 3.6 ug/bee, while the 24-hour and 48-hour LC_{50} for the amphipod *Gammarus lacustris* were 10 ppm and 5.8 ppm, respectively (Pimentel 1971).

Vertebrates--The 24-hour and 48-hour LC_{50} of dicamba for juvenile coho salmon (*Oncorhynchus kisutch*) were 151 ppm and 120 ppm (Bond et al. 1965) but for rainbow trout (*Salmo gairdneri*) and bluegills (*Lepomis macrochirus*) the 48-hour LC_{50} were 35 and 130 ppm, respectively (Pimentel 1971). The acute oral LD_{50} in pheasants (*Phasianus colchicus*) was 673 mg/kg in females and 800 mg/kg in males (Pimentel 1971). The acute oral LD_{50} in male rats was 757 mg/kg, with 95 percent confidence limits of 449 to 1276 mg/kg while the acute oral LD_{50} of formulated dicamba to male rats was 1100 mg/kg with 95 percent confidence limits of 925 to 1308 mg/kg (Edson and Sanderson 1965).

Khristov (1975) noted that dicamba at concentrations of 0.1 to 10 ppm was mutagenic in corn and caused chromosome damage. No data on its mutagenesis in animals or bacterial systems were available. Levels of 800 ppm for three months, or 500 ppm for two years, were not overtly toxic to rats or dogs, and three generations of rats fed dicamba were reproductively normal (USDA 1974).

Conclusions:

Although dicamba is not a highly persistent herbicide, it can persist for long periods under certain conditions. Moreover, its metabolite, 3,6-dichlorosalicylic acid, may be considerably more persistent than dicamba itself. Dicamba cannot be recommended for soil disposal until more is known of its terminal residues.

Diquat and Paraquat

Diquat, the common name for the 6,7-dihydro (1,2-*a*:2',1'-*c*) pyrazinedium ion, is the active ingredient of diquat dibromide, introduced by I. C. I. Plant Protection Ltd. in 1958 as Reglone. It is marketed as Ortho Diquat by Chevron Chemical Co. in the United States. Diquat dibromide monohydrate, a yellow solid which decomposes above 300°C without melting, is highly soluble in water (700,000 ppm) and is corrosive to most common metals. It is a contact herbicide which is highly susceptible to decomposition by ultraviolet light but essentially nonvolatile. It is synthesized by condensing 2,2'-dipyridyl with ethylene dibromide.

Paraquat is the common name for 1,1'-dimethyl-4,4-bipyridinium ion, the active ingredient of the contact herbicide paraquat dichloride, which was introduced by I. C. I. Plant Protection Ltd. in 1958 as Gramoxone. It is marketed in the U. S. by Chevron Chemical Company under the name Ortho-Paraquat. Pure paraquat dichloride is a white crystalline solid with a faint ammoniacal odor, which decomposes at about 300°C. It is completely soluble in water and is formulated only in aqueous solution. Technical paraquat dichloride is a dark red solution, which is corrosive to most common metals. It is synthesized by direct quaternization of 4,4'-bipyridyl with methyl chloride under pressure (WSSA 1974).

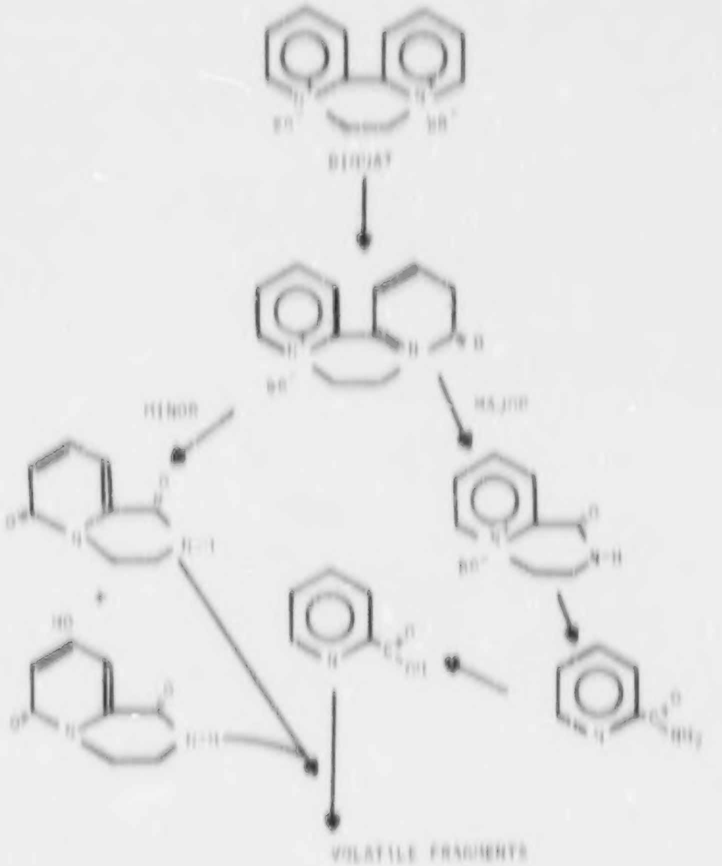
Degradation-

Biological--Both diquat and paraquat are degraded by microorganisms. Atkinson (1973) reported an *Achromobacter* which reduced diquat and Hamdeo (1972) isolated nine actinomycetes, one *Streptomyces* and one *Nocardia* capable of decomposing paraquat with 1-methyl-4-carboxypyridylium ion as a degradation product. Funderburk and Bozarth (1967) also found a bacterium capable of degrading paraquat by demethylation followed by ring cleavage. The 1-methyl-4-carboxypyridinium ion was identified as a product.

Picolinamide, a photolytic product of diquat, is degraded by bacteria (Menzie 1974). The degradation of the 4-carboxy-1-methyl-pyridinium ion by *Achromobacter D* and that of *N*-methylisonicotinate by a gram-positive and a gram-negative bacterium have also been shown (Menzie 1974). Microorganisms have been cultured which can use paraquat as their sole source of nitrogen (Funderburk 1966). These data make it apparent that, at least in the laboratory, microbial degradation of diquat and paraquat is plausible. In soil under field conditions, adsorption radically reduces the plausibility of microbial decomposition (Weber and Coble 1968). Fryer and co-workers (1975) concluded that paraquat may not break kown at all in soil, and that if microbial degradation occurs, it is extremely slow. Funderburk and Bozarth (1967) stated that both herbicides are quite stable in higher plants.

In animals, bipyridylium compounds are poorly absorbed from the gut and are excreted, unchanged, in the feces. Paulson (1975), reviewing the fate of herbicides in animals, states that partial metabolism occurs, but that the nature of the products has not yet been reported. The role of microorganisms in the gut in metabolizing diquat and paraquat has also not been established.

Photolytic--Dry paraquat and diquat were 50 percent decomposed by UV light in 48 hours. No diquat remained in an aqueous solution exposed to ultraviolet light for 192 hours (Funderburk and Bozarth 1967). In aqueous solution, paraquat was totally decomposed within three days to the *N*-methylbetaine of nicotinic acid, with subsequent formation of methylamine hydrochloride and nicotinic acid (Slade 1965). The 1-methyl-4-carboxypyridinium ion was obtained from paraquat in aqueous solution exposed to 240-260 nm UV light. On planchets, inactivation was 50 percent completed in 48 hours, and 75 percent completed in 96 hours (Funderburk et al. 1966). The photolytic degradation of diquat is shown in Figure 3.


DIQUAT
MINOR
MAJOR
VOLATILE FRAGMENTS

PHOTOLYTIC DEGRADATION OF DIQUAT
(AFTER MENZIE 1974)

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Figure 3

Chemical And physical--Hance (1967) considered the chemical degradation of paraquat on soil to be extremely slow due to adsorption and relatively low temperatures. In water, at pH = 8, 0.03 mg/ml paraquat was oxidized by ClO_2 within one minute (Gomaa and Faust 1971) and at neutral or acid pH, paraquat is very stable (Hiltibran *et al*. 1972, WSSA 1974). Paraquat was not at all decomposed by treatment with 100 percent methanolamine (Kennedy *et al*. 1972b) but was 99.9 percent decomposed by either metallic sodium or lithium with liquid ammonia and 95 percent was decomposed by sodium biphenyl (Kennedy *et al*. 1972a). At 600°C, 98.3 percent of paraquat in commercial formulations decomposed (Kennedy *et al*. 1969). Volatile products of burning analytical-grade paraquat at 900°C in a muffle furnace were carbon monoxide, carbon dioxide, and ammonia (Kennedy *et al*. 1972b).

Transport-

Within soil--The major factor determining the mobility of paraquat and diquat in soil is their adsorption by clay and by organic matter (Burns and Hayes 1974, Khan 1974, Hayes *et al*. 1975). The adsorption of the bipyridylium ions on montmorillonite is generally considered irreversible under field conditions (Tomlinson *et al*. 1968, Weber *et al*. 1965, Gamar and Mustafa 1975, Nithyantha and Perur 1975). The complete recovery of bipyridylium herbicides from soil required four hours of refluxing with 18N sulfuric acid (Tucker *et al*. 1967). Adsorption was a function of the cation exchange capacity of the clays (Malquori and Radaelli 1966, Weber *et al*. 1965) and charge-transfer complexes are apparently formed between diquat or paraquat and soil organoclay complexes (Khan 1973). Tucker *et al*. (1967) recognized two types of binding between soil and bipyridilium herbicides: tight and loose, the former irreversible and the latter reversible when the soil was leached with concentrated salt solutions. The ratio of tightly bound to loosely bound paraquat or diquat was 1:4 in loam, 1:27 in sand and 1:107 in muck (Tucker *et al*. 1967). Adsorption to kaolinite, but not to montmorillonite, could be reversed by liming or fertilizing (Weber *et al*. 1965) and desorption from bentonite was easier than from muck (Harris and Warren 1964). Adsorption of paraquat was greater on acid than neutral soils (Weber and Best 1972). On humic acid or humin, paraquat was more strongly adsorbed to strongly acidic sites, but diquat to weakly acidic sites (Best *et al*. 1972). The relative adsorbability of the dipyridylium cations on organic matter was: Ca^{++} - humus > H^{+} - humus > lignin > epidium roots > oats > clover, lucerne or wheat (Radaelli and Fusi 1968).

Unlike most pesticides, diquat and paraquat directly influence the stability of the soil, due to the dipyridyl cation, particularly in clay soils with few water-stable aggregates (Maiquori and Radaelli 1968). Gomar and Mustafa (1975) calculated that 1,930 tons per acre of diquat, and up to 4,500 tons per acre of paraquat, could be adsorbed by arid-zone soils and that less than one percent of such adsorbed herbicide could be released by water. Such an enormous and tenacious capacity of adsorption is, as expected, correlated with an extremely low mobility (Helling 1971a). Nevertheless, paraquat applied to a sandy-loam soil was found in significant amounts at the 25 to 35 cm level (Fryer *et al*. 1975).

Between soil and water--Since paraquat and diquat are frequently used as aquatic herbicides, contamination of water by direct application is most common. Calderbank (1972) noted that diquat and paraquat were adsorbed by mud after the aquatic weeds died, and that no desorption from mud occurred. After six months, 36 percent of the paraquat applied to the water remained in the top two inches of hydrosoil, while water levels fell from 2.5 ppm to 0.01 ppm within two weeks (Calderbank 1970). Atkinson (1973) estimated that free diquat persisted no more than two hours in water, being adsorbed by sediment thereafter. In reservoirs, 74 percent of diquat and paraquat were adsorbed by sediment within half an hour (Yeo 1967). As Hiltibran and co-workers (1972) suggested, the hydrosoil may well be the ultimate repository of diquat, and probably also of paraquat.

Into organisms--Diquat and paraquat are taken up by plants to some extent even though their herbicidal activity is by contact (WSSA 1974). Water levels of one ppm led to 112 ppm paraquat in the aquatic plant *Myriophyllum spicatum* in two days (Calderbank 1972). Cattle fed 200 to 400 ppm paraquat did not secrete it in their milk or muscles, and fish accumulated paraquat only in skin, gills and viscera, not in their edible tissues (Calderbank 1972). Rainbow trout exposed to [illegible] ppm did not concentrate paraquat in their tissues (Calderbank 1970).

Persistence--

Paraquat does not retain its phytotoxicity when adsorbed to clay, and retains very little when adsorbed to soil organic matter. Phytotoxic persistence is, therefore, not very great (WSSA 1974). In an orchard soil in Ontario 2.24 kg/ha paraquat applied in early June each year from 1963 to 1971 led to residues of 5.36 ppm in the top 15 cm of soil in July of 1971, and 4.12 ppm 16 weeks later (Khan et al. 1975). This constituted approximately 50 percent of the total 20.16 kg/ha applied over the nine-year period--a remarkable persistence for a foliage-applied herbicide subject to photolysis. Fryer, Hance and Ludwig (1975) found that essentially all the paraquat applied to a sandy loam soil remained in the soil for two years, and suggested that paraquat is not degraded at all in soil.

Water levels of diquat decreased from 1,000 ppb to nine ppb in 12 days in reservoirs, and paraquat applied at 250-2,500 ppb declined to less than 180 ppb in 13 days (Yeo 1967). In some New York lakes, four lbs/A (4.48 kg/ha) of diquat applied to water declined to less than 0.005 ppm within eight days (Sewell 1970). Most of the dissipated paraquat and diquat are merely absorbed to hydrosoil according to Hiltibran et al. (1972) and Frank (1967).

Effects on Non-Target Species:

Microorganisms--Paraquat and diquat are only toxic to soil processes and soil microorganisms at very high levels *in situ*, probably because they are so completely inactivated by clay as well as by soil organic matter. Of nine herbicides tested, only dichlobenil was less toxic to ammonia oxidation than paraquat: picloram, 2,3,6-TBA, Chlorthiamid, bromoxynil, chlorfluorazole, ioxynil and propanil were more inhibitory (Debona and Audus 1970). The effects of diquat on soil processes and soil microorganisms are shown in Tables 13 and 14.

Overall bacterial numbers were decreased by 500 ppm of paraquat (Pokorny 1971). Proteolytic bacteria were inhibited for four days, beginning two days after treatment, by 400 ppm paraquat, while cellulolytic bacteria were stimulated for eight days, inhibited between the 8th and 10th day, and then returned to normal levels. Even at 400 ppm, paraquat had no effect on ureolytic bacteria (Giardina *et al.* 1973). Overall bacterial populations were depressed for at least 56 days by five tons/acre (5,000 ppm) of paraquat, whether pure or formulated (Stojanovic *et al.* 1972a) but not by low levels (Debona and Audus 1970), and 5.6 kg/ha were even stimulatory (Camper *et al.* 1973).

Bacterial species varied widely in the levels of paraquat required for inhibition, from 10^{-6} mg/liter (one ppm) for *Azotobacter chroococcum* in a nitrogen-free medium (Manninger *et al.* 1972) to one mg/ml (1,000 ppm) for *Bacillus subtilis* (Thomas *et al.* 1973). One *Bacillus* species was inhibited by ten liters of Gramoxone per hectare (Langkramer 1970) and another by five ppm (Breazeale and Camper 1972). Paraquat inhibited the growth of *Pseudomonas fluorescens*, *Erwinia carotovora*, and *Escherichia coli* at 25, 30 and 50 ppm, respectively (Breazeale and Camper 1972, Wallnoefer 1968).

Inhibition of fungi was reported at 56 ppm and stimulation at 2.8 ppm paraquat (Camper *et al.* 1973). Stojanovic *et al.* (1972a) stimulated

Table 13. EFFECTS OF DIQUAT ON SOIL PROCESSES

Parameter	Effect	Level		Conditions	Reference
		Original	PPM		
Respiration	–			Montmorillonite in nutrient sol'n	Weber and Coble 1968
	0			Kaolinite in nutrient sol'n	Ibid.
Ammonification	–	5 ppm	5	by *Desulfovibrio*	Atkinson 1973
		100 ppm	100	by *Pseudomonas*	Ibid.
Cellulolysis	–			heavy soils	Rankov 1970
Nitrogen fixation	–	≤5 ppm	≤5	by *Clostridium pasteurianum*	Atkinson 1973
	–	≤5 ppm	≤5	by *Rhodospirillum rubrum*	Ibid.
Nitrification	–	≤20 ppm	≤20	by *Nitrosomonas*	Ibid.
Proteolysis	–	≤20 ppm	≤20	by bacteria	Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 14. EFFECTS OF DIQUAT ON MICROORGANISMS

Parameter	Effect	Level		Conditions	Reference
		Original	PPM		
*Microorganisms	-	50 mg/l	50		Pokorny 1971
Bacillus sp.	-	25 µg/ml	25		Breazeale and Camper 1972
Erwinia carotovora	-	25 µg/ml	25		Ibid.
Bacillus subtilis	-	1 mg/ml	1000		Thomas *et al.* 1973
Pseudomonas	-	<200 ppm	<200		Wallnofer 1968
Escherichia coli	-	25 ppm	25		Ibid.
Aerobacter	-	<200 ppm	<200		Ibid.
Aspergillus niger	-	<200 ppm	<200		Ibid.
Trichoderma viride	-	<200 ppm	<200		Ibid.
Rhizopus japonicus	-	<200 ppm	<200		Ibid.
	-	<200 ppm	<200		Ibid.
algae	-	20 ppm	20	less than 28 days	DaSilva *et al.* 1975
Chlorella pyrenoidosa	-	>0.1 mg/ml	>100	paper disc culture	Thomas *et al.* 1973

+ = stimulation
0 = no effect
- = inhibition

Streptomyces by incorporating 5,000 ppm pure paraquat into loam, but formulated paraquat at levels of 5,000 ppm inhibited *Streptomyces*. Of thirteen fungal species tested by Szegi (1973), all except four *Fusarium* species were depressed by 25 ppm of paraquat. The inhibited species were: *Aspergillus candidus*, *A. ustus*, *Stachybotrys atra*, *Verticillium candelabrum*, a *Penicillium* species, and two species of *Fusarium*. *Fusarium oxysporum* was not inhibited by 20 ppm (Rodriguez-Kabana and Curl 1972) but *Suillus variegatus* was inhibited by 2.3 ppm (Sobotka 1970).

These data make it abundantly clear that, even though paraquat does not depress soil respiration at five tons per acre (5,000 ppm) (Stojanovic *et al.* 1972a), the balance among soil microflora will be sharply altered as sensitive species are replaced by resistant species. Cellulolytic bacteria are inhibited by paraquat levels of 500 ppm or more (Szegi 1973, Szegi and Gulyas 1971). Rankov (1970) considered paraquat to have no effect on cellulolytic bacteria in soil, but did not specify levels.

The effects of paraquat on aquatic systems are extreme: 0.6 ppm was toxic to freshwater bacteria, freshwater algae and freshwater protozoa (Huess 1972). Bluegreen algae were affected for up to 28 days by 10 ppm (Da Silva *et al.* 1975) and 100 ppm was toxic to *Chlorella* on paper disc culture (Thomas *et al.* 1973).

Molds were less sensitive to diquat and paraquat than were bacteria (Wallnoefer 1968). In pure cultures, ten to 100 ppm inhibited growth of *Trichoderma viride* and *Rhizopus stolonifer* but not of *Fusarium culmorum* or *Aspergillus niger*. In mixed cultures, both paraquat and diquat reversed the usual dominance of *R. stolonifer* over *A. niger* (Wilkinson 1969). On agar, 100 ppm paraquat resulted in selection for resistant bacteria (Kokke 1970a) and resistance was observed in fungi from paraquat-treated soil (Szegi and Gulyas 1971). Gene conversion was induced in *Saccharomyces cerevisiae* by 100 to 900 ppm paraquat (Hamdeo and Dube 1973). Rosival (1970) included both diquat and paraquat among the pesticides exerting direct chemical effects on DHA.

Invertebrates--Even at one ppm diquat affected aquatic arthropods and molluscs, albeit indirectly, through habitat destruction (Hilsenhoff 1966). In a New Zealand stream, three 30 minute exposures to two ppm of paraquat destroyed 95 percent of the amphipods within days, but increased the numbers of *Trichoptera* after one year (Burnett 1972). The 96-hour TL_M of diquat for pond invertebrates ranged from 0.048 ppm in the amphipod *Hyalella azteca* to over 100 ppm for dragon flies (*Libellula*) and damselflies (*Enallagma*) (Wilson and Bond 1969). No short-term effect on aquatic macro-invertebrates was incurred when 0.11 ppm diquat and 0.17 ppm endothall were used (Berry et al. 1975).

The toxicity of paraquat to entomophagous mites and insects was considered too great to permit its use in an integrated pest control program (Kock and Yeargan 1973). Under laboratory conditions, the relative toxicities of eight herbicides to beetles (*Bembidion*) were: sodium chlorate < dalapon < simazine < simazine plus amitrole < pyrazon < diquat < 2,4-D < chlorpropham (Mueller 1971).

Fish and amphibians--The 96-hour LC_{50} of diquat to striped bass (*Roccus saxatilis*) in aquaria was 0.25 ppm (Wellborn 1969) while the TL_M of paraquat for bluegills (*Lepomis macrochirus*) was 400 ppm at 24 hours and 100 ppm at 48 hours (Davis and Hughes 1963). Gilderhus (1967) found that bluegills accumulated increasing amounts of diquat for ten days after water treatment and that residues persisted for six to twelve weeks. At high concentrations (275 ppm), diquat increased the time of onset for the toxicity of aldrin, DDT or parathion without decreasing its severity (Krieger and Lee 1973). Despite the indubitable toxicity of diquat and paraquat for fish, Schluster (1970) considered it a suitable aquatic herbicide if carefully handled. Hiltibran (1967) considered the no-effect level of diquat for bluegill eggs and fry to be ten ppm and concluded that, at ordinary levels of application, diquat would not seriously harm fish. The 24-hour LC_{50} of paraquat for Fowlers toad tadpoles (*Bufo woodhousii fowleri*) was 54 ppm (Pimentel 1971).

Birds--The five day LC_{50} of diquat for two-week old quail was 1,346 mg/kg with 95 percent confidence limits of 1,178 to 1,540 mg/kg; 3,742 mg/kg with 95 percent confidence limits of 3,329 to 4,220 for pheasants; 2,933 mg/kg with 95 percent confidence limits of 1,181 to 5,256 mg/kg for bobwhites; and mallard mortality was 30 percent at 5,000 mg/kg. For paraquat, the five day LC_{50} was 970 mg/kg (823-1,140 mg/kg) for quail; 981 (784-1,213 mg/kg) for bobwhite; 1,468 (1,287-1,673) mg/kg for pheasants; and 4,048 (3,432-4,886 mg/kg) for mallards (Heath et al. 1972).

When diquat was injected into fertilized hens' eggs, ten percent hatched if 0.5 mg (ten ppm) were injected and 60 percent hatched after five ppm were injected (Dunachie and Fletcher 1967). Paraquat was lethal to over half the chick embryos even at 0.15 ppm injected on the first day of incubation. One ppm permitted survival of 25 percent of embryos treated on day two, and of 94 percent treated on day 16 (Dunachie and Fletcher 1970). At 0.1 ppm injected, egg hatch was as high as in controls (Dunachie and Fletcher 1967). Fletcher (1957) considered the levels injected into the eggs to correspond to feeding the hens 40 ppm of paraquat.

Mammals--The acute oral LD_{50} of paraquat in rats was 100 to 110 mg/kg with 95 percent confidence limits between 85 and 134 mg/kg (Gaines 1969). Howe and Wright (1963) cited 200 mg/kg paraquat and 400 mg/kg diquat in rats and 40 to 50 mg/kg paraquat in cats. The no-effect levels for dogs fed paraquat or diquat for three years was 7.5 mg/kg, a level rats could tolerate over their whole life span. Paraquat reportedly altered the fight-flight response of rats at levels above five percent of the LD_{50} (Billewicz-Stankiewicz and Pawlowski 1971). Both diquat and paraquat affected spermatogenesis, but not preimplantation mortality, in rats (Pasi et al. 1974).

Conclusions-

The absolute lack of evidence for any degradation of paraquat or diquat in soil makes these compounds unsuitable for soil disposal. Use of sunlight to decompose these herbicides in water might be possible if contact with adsorptive surfaces such as hydrosoil is prevented. Pyrolysis is also a feasible method of disposal.

SUBSTITUTED UREA HERBICIDES

Diuron, Linuron, Monuron and Monolinuron

The urea herbicides monuron [*N'*-(4-chlorophenyl)-*N*-methoxy-*N*-methylurea], diuron [*N'*-(3,4-dichlorophenyl)-*N*,*N*-dimethylurea] and linuron [3-(3,4-dichlorophenyl)-1-methoxy-1-methylurea] were introduced by E. I. duPont de Nemours in 1951, 1955 and 1960, respectively. Monolinuron [*N'*-(4-chlorophenyl)-*N*-methoxy-*N*-methylurea] was introduced by Farbwerke Hoechst AG in 1960. All are used as selective herbicides to some extent, but monuron and diuron are used primarily for weed control in uncropped areas, while linuron and monolinuron are primarily preemergent herbicides.

Monuron is synthesized by the reaction of 4-chlorophenyl isocyanate with dimethylamine and diuron is synthesized by reacting 3,4-dichlorophenyl isocyanate with methylamine. Linuron is prepared by the reaction of 3,4-dichlorophenyl isocyanate with *O*,*N*-dimethyl hydroxylamine, whereas monolinuron is synthesized by the reaction of hydroxylamine and 4-chlorophenyl isocyanate followed by reaction with dimethyl sulfate. The characteristics of each compound are shown in Table 15.

Degradation-

Biological--The substituted urea herbicides are decomposed by many soil organisms to some extent. Breakage of the bond between the carbonyl carbon and nitrogen atoms absolutely requires a urease, but methyl dealkylation is far less specifically catalyzed (Kearney 1966). Boerner (1967) noted that demethylation is carried out by numerous microorganisms. The initial products consist of methyl- and methoxyureas, chlorinated anilines, and carbon dioxide derived from the side chains (Tables 16 and 17). Boerner (1967) reported that 3,4-dichloroaniline was also rapidly degraded in loamy soil but Menzie (1974) considered aniline formation to be of some concern.

Table 15. PHYSICAL CHARACTERISTICS OF MONURON, DIURON, LINURON AND MONOLINURON

Characteristic	Monuron	Diuron	Linuron	Monolinuron
Color	white	white	white	white
Odor	none	none	none	none
Melting point	174-175°C	158-159°C	93-94°C	79-80°C
Decomposition	185-200°C	180-190°C	-	-
Vapor pressure	5×10^{-7} mm Hg at 25°C	3×10^{-6} mm Hg at 50°C	1.5×10^{-5} mm Hg at 24°C	1.5×10^{-4} mm Hg at 22°C
Water solubility	230 ppm	42 ppm	75 ppm	580 ppm
Agri. levels	5.4-34 kg/ha	5.6-37.2 kg/ha	0.9-1.1 kg/ha	0.6-7.2 kg/ha

Table 26. DEGRADATION OF MONURON AND DIURON BY SOIL MICROORGANISMS

Organism	Products	Conditions	Reference
Monuron			
Aspergillus niger *Aspergillus sydowi* *Aspergillus tamarii*	unidentified	5,10,20 ppm in sand	Murray *et al.* 1969
Bacillus sphaericus			Engelhardt *et al.* 1971
Fusarium; *Botrytis*	CO_2; unidentified metabolites	500 ppm in sandy loam for 80 days	Kaufman and Blake 1973
Rhizopus japonicus	3-(p-chlorophenyl)-1-methylurea		Wallnoefer *et al.* 1973
Pseudomonas or *Arthrobacter*	3,4-dichloroaniline, sym-bis(3,4-dichlorophenyl)urea, unknowns		Jasko *et al.* 1970
Not identified	3-(3,4-dichlorophenyl)-1-methylurea; 1-(3,4-dichlorophenyl) urea; 3,4-dichloroaniline	soil in mid-South	Dalton *et al.* 1966
Aspergillus ustus	3% degradation in 7 days	soil	Kaufman and Blake 1973
Fusarium oxysporum	7.4% degradation in 20 days	soil	Ibid.
Achromobacter	4.1% degradation in 20 days	soil	Ibid.
Aspergillus niger *Aspergillus sydowi* *Aspergillus tamarii*	unidentified	5,10,20 ppm in sand	Murray *et al.* 1969

Table 17. DEGRADATION OF MONOLINURON AND LINURON BY SOIL MICROORGANISMS

Organism	Products	Conditions	Reference
Monolinuron			
Unidentified	3,4-chloroaniline	soil	Boerner 1965
Aspergillus niger	phenylmethoxyurea, phenylmethylurea, chloroaniline, CO_2, NH_3	soil	Boerner 1967
Bacillus sphaericus	4-chloroaniline, CO_2, unknowns		Engelhardt et al. 1971, Wallnoefer 1969, Wallnoefer and Bader 1970, Wallnoefer and Engelhardt 1971
Bacillus sphaericus	N,O-dimethylhydroxylamine	sandy loam soil	Schuphan 1974
Rhizopus japonicus	3-(p-chlorophenyl)-1-methoxyurea		Wallnoefer et al. 1973
Unidentified	0.4% 3-methoxy-3'-4-chlorophenylurea	waste composting	Mueller and Korte 1975
Linuron			
Unidentified	3,4-chloroaniline	soil	Boerner 1965
Aspergillus niger	phenylmethoxyurea, phenylmethylurea, CO_2, NH_3, chloroaniline	soil	Boerner 1967
Bacillus sphaericus	N,O-dimethylhydroxylamine, CO_2		Engelhardt et al. 1972

Bacillus sphaericus degraded monolinuron and linuron but not monuron or diuron (Engelhardt et al. 1971). The decomposition consisted of removal of the ureido portion of the molecule from *N*-methoxy-compounds with formation of *p*-chloroaniline and the latter was reportedly resistant to further degradation (Wallnoefer 1969, Wallnoefer and Bader 1970, Wallnoefer and Engelhardt 1971). Wallnoefer et al. (1973) reported that *Rhizopus japonicus* dealkylated both monuron and monolinuron. Mueller and Korte (1975) observed that while some monolinuron was demethylated during waste composting, 86.2 percent was unaltered. Kaufman and Blake (1973) cultured ten microorganisms which decomposed propham, propanil, solan, and SWEP, of which three (*Aspergillus ustus*, *Fusarium oxysporum* and *Achromobacter*) also degraded diuron. *Aspergillus versicolor*, three strains of *Penicillium*, *Fusarium solanum* and *Trichoderma viride* did not decompose diuron. Lopez and Kirkwood (1974) obtained growth, but no diuron degradation, when *Fusarium* and two bacteria were cultured with 20 ppm diuron for two weeks. After 80 days, 3.5 percent of the radioactive label evolved as $^{14}CO_2$ and increasing the soil temperature by 10°C between 10° and 30° tripled diuron degradation. Of ten ppm diuron, 6.3 ppm remained after 12 weeks in loamy sand, 5.5 ppm after 12 weeks in clay loam (McCormick and Hiltbold 1966).

Murray and co-workers (1969) compared the degradation of five substituted urea herbicides by three strains of *Aspergillus*. The herbicide fenuron, with no chlorine, was most readily decomposed by all three strains, while diuron was most resistant. Norea, fluometron and monuron were intermediate. Some growth was achieved by *A. sydowi* and *A. tamarii* even at 1000 ppm of monuron, but *A. niger* was completely inhibited by 100 ppm. Rankov (1968b) found that 0.01 percent monolinuron (100 ppm) served as nitrogen source for five of 25 strains of soil bacteria, and ten of 20 strains of actinomycetes, while 100 ppm of linuron served as nitrogen source for five of 25 bacteria, but only two of twenty actinomycetes. Five of 15 strains of fungi could use linuron or monolinuron as a nitrogen source. Maximum degradation of monolinuron occurred when soil water levels were at 30 percent of the soil's water capacity and after repeated applications (Suess 1970). Belasco and Pease (1969) examined soil after 12 years of annual diuron treatment (2.24 and 4.8 kg/ha) or after one year of linuron treatment (2.24 kg/ha) and found no 3,3',4,4'-tetrachloroazobenzene, while incubation of 500 ppm linuron or diuron resulted in formation of about one ppm of dichloroaniline and no 3,3',4,4'-tetrachloroazobenzene. As with most pesticides, increasing levels of application decreased the rate of degradation (Hance and McKone 1971).

In sterile soil, $^{14}CH_3O$-labeled monuron decomposed to form nine percent $^{14}CO_2$, and 28 percent *N,O*-dimethylhydroxylamine, but in nonsterile soil $^{14}CO_2$ accounted for 58 percent of the radioactivity and *N,O*-dimethylhydroxylamine for only eight percent (Schuphan 1974a, 1974b). The decomposition of monolinuron to *N,O*-dimethylhydroxylamine could, at least theoretically, lead to nitrosamine formation (Schuphan 1974b). In spinach, monolinuron was metabolized to 3-(4-chlorophenyl)-1-methoxy-1-hydroxymethylurea (Schuphan and Ebing 1975).

The fate of urea herbicides in animals has been reviewed by Paulson (1975) who concluded that the urea moiety is hydrolyzed only to a limited extent, and that the major routes of metabolism include demethylation, demethoxylation, ring hydroxylation and conjugation. Boehme and Ernst (1965a, 1965b) fed diuron to rats and found that urinary metabolites accounted for about 20 percent of the dose. Metabolites included 1-(3,4-dichlorophenylurea) and 1-(2-hydroxy-4,5-dichlorophenyl)urea as a major urinary products. The tissues and feces were not analyzed.

Photolytic--Diuron was decomposed by UV light (Janko et al. 1970). Monuron and diuron were decomposed by both UV light or sunlight, but different products resulted from the two kinds of light (Jordan et al. 1964). Weldon and Timmons (1961) noted that most decomposition of diuron occurred between 227 nm and 249 nm, while for monuron most changes occurred between 220 nm and 245 nm. Exposure to 28 hours of UV light reduced the phytotoxicity of 0.5 lb/A (0.56 kg/ha) diuron from 30 percent kill of oat plants to zero, and of monuron from 100 percent kill to zero.

The photolytic decomposition of monuron exposed to southern California sun for 14 days were 3-(*p*-chlorophenyl)-1-formyl-1-methyl-urea, 1-(*p*-chlorophenyl)-3-methylurea, 4,4'-dichlorocarbanilide, 3-(4-chloro-2-hydroxyphenyl)-1,1-dimethylurea. Three additional products were tentatively identified: 1-(*p*-chlorophenyl)-3-formylurea, 4'-chloroformanilide, and *p*-chloroaniline. The degradation proceeded by stepwise photo-oxidation and demethylation of the *N*-methyl groups, hydroxylation of the aromatic nucleus, and polymerization. Degradation was hydrolytic and the products formed were not dependent on the initial concentrations of monuron. Less than six percent of the monuron was decomposed after 14 days, and little HCl was formed (Crosby and Tang 1969). Mazzochi and Rao (1972) photolysed monuron in methanol at 253.7 nm, under anaerobic conditions. The products were 3-phenyl-1,1-dimethylurea and methyl-*p*-chlorophenylcarbamate, the latter in minor amounts.

Linuron exposed to sunlight for two months decomposed to 3-(3-chloro-4-hydroxyphenyl)-1-methoxy-1-methylurea, (13 percent), 3,4-dichlorophenylurea (ten percent) and 3-(3,4-dichlorophenyl)-1-methylurea (two percent), while 69 percent of the linuron remained unchanged (Rosen *et al.* 1969).

Chemical and physical--Linuron decomposed at 95°C (Janko *et al.* 1970). Extrapolating from the decomposition of diuron and linuron at temperatures of 85°C or higher, Hance (1967) estimated that their nonbiological degradation in soil would require more than nine years, but when the effects of soil adsorption were included, the model suggested that nonbiological degradation in soil was plausible (Hance 1969). The decomposition of diuron in soil was not affected by pH (Corbin and Upchurch 1967).

The bioactivity of 100 ppm diuron in water exposed to three Mrads of *gamma*-irradiation was less, but not significantly less, than that of 100 ppm diuron stored for the same length of time without irradiation. In the solid state, diuron was not decomposed by three Mrads of *gamma* irradiation (Horowitz and Blumenfeld 1973). Diuron was completely decomposed by liquid ammonia in the presence of metallic sodium, and 96.8 percent by liquid ammonia in the presence of lithium (Kennedy *et al.* 1972a). At 775^{0}C, 25 percent of technical diuron was decomposed whereas diuron formulated as an 80 percent wettable powder was 25 percent decomposed at 550^{0}C (Kennedy *et al.* 1969). Complete combustion at 900^{0}C produced carbon monoxide, carbon dioxide, chlorine, hydrogen chloride, ammonia, and nitrogen oxides as volatile products (Kennedy *et al.* 1972a, 1972b).

Transport:

Within soil--Spiridonov and co-workers (1970) reported that monuron and diuron leached 100 cm during 110 days in moist subtropical soil. Liu (1974) reported that diuron leached 36 inches (90 cm) during 112 days in a Puerto Rican soil, and Dowler *et al.* (1968) reported 170 cm leaching in 90 days in Puerto Rican forests. More typically, diuron and monuron leached 20 to 30 cm in 150 days (Spiridonov and Yakovlev 1967) or 45 cm in 180 days (Leh 1968). At ten kg/ha, monuron and diuron leached 40 to 50 cm, while 30 kg/ha leached 60 to 70 cm (Spiridonov *et al.* 1968). For diuron, Ivey and Andreva (1965) reported six inch (15 cm) soil penetration of agricultural levels, but eight inch (20 cm) penetration of 2.5 times agricultural levels. Kazarina (1965) found that monuron applied to soil columns leached more readily in loamy sand than in sandy loam. Khubitiya and Giginelshvili (1971) observed 40 cm leaching of monuron during a 60 day period in early spring.

Cool dry climates were said to inhibit both leaching and decomposition of monuron (Erickson 1963). Granules were less mobile than other formulations (Spiridonov *et al.* 1968) and diuron granules remained in the surface layers of soil when a two to three cm layer of water was on the soil (Imaliev and Beresnova 1969). Liu (1974) estimated that 3.6 percent of the diuron applied to Puerto Rican Vega Alta soil was lost to leaching, with the greatest transport during the first week and no observable leaching after 16 weeks. One year after the third of three annual applications of monuron, 62 to 69 percent of the residues recovered remained in the top two inches of soil, and 86 to 100 percent remained in the top four inches (Dawson *et al.* 1968).

The substituted urea herbicides are moderately mobile, with relative mobilities of 2.2 to 3.3 on a scale of six for monuron, diuron and linuron (Harris 1967). The precise degree of leaching depends, as always, on numerous factors among which are water flux and average pore velocity (Davidson and Santelman 1968, Davidson *et al.* 1968), electrolytes (Hurle and Freed 1972) and the presence of surfactants (Bayer 1967). Adsorption by soil is the major deterrent to leaching, and all the urea herbicides are readily absorbed by organic matter (Harris and Warren 1964, Doherty and Warren 1969, Hilton and Yuen 1963, Liu *et al.* 1970, MacNamara and Toth 1970, Moyer *et al.* 1972, Green and Young 1971, Mustafa and Gamar 1972). Adsorption to clay is also significant (Scott and Lutz, 1971, Harris and Warren 1964, Doherty and Warren 1969, Khan 1974, MacNamara and Toth 1970, Moyer *et al.* 1972, Mustafa and Gamar 1972, Van Bladel and Moreale 1974) as is adsorption to carbon (Hilton and Yuen 1963, Jordan and Smith 1971). Models for the mobility and adsorption of the urea herbicides have been constructed frequently (Rhodes *et al.* 1970, Khan and Mazurkewich 1974, Huggenberger *et al.* 1973, Hance 1969b, Aleshin and Yudina 1973).

Cation exchange capacity and specific surface area affect adsorption (Mustafa and Gamar 1972, Doherty and Warren 1969, Liu *et al.* 1970, MacNamara and Toth 1970). On montmorillonite, adsorption depended on the ionic strength of the saturating solution at high ionic strengths (Van Bladel and Moreale 1974). In dry soils, linuron formed a stable complex with montmorillonite (Khan 1974). Adsorption of monuron on bentonite increased with increasing temperature from 0° to 50°C, but adsorption on muck was not significantly affected by temperature. Desorption of monuron from bentonite occurred more readily than from muck (Harris and Warren 1964). Diuron adsorption on Puerto Rican soils decreased linearly with increasing temperatures (Liu *et al*. 1970).

Relative adsorption of linuron by organic matter and clay was: muck and peat > sphagnum moss ≥ bentonite ≥ quartz sand (Doherty and Warren 1969). Monuron was reversibly adsorbed to peat if and only if the peat was not dried, while linuron could never be completely desorbed from peat (Moyer *et al*. 1972). Harris (1966) found that monuron was adsorbed least by Lakeland sandy loam, most by Chillum silt loam or Hagerstown silty clay, with Wehadkee silt loam intermediate; but monuron was mobile in all four soils.

Monuron and diuron leached more in alluvial sandy soil than in brown forest soil (Khubutiya and Gigineishvili 1971). Increased moisture levels, whether from rain or subirrigation, increased monuron transport in soil columns containing loamy sand or sandy loam (Kazarina 1965).

Hance (1965a, 1965b, 1969a, 1969b, 1974) and Grover and Hance (1970) examined the adsorption of urea herbicides by soils. Among substituted urea derivatives, adsorption increased with increasing chain length and with addition of chlorine or chloro-phenoxy-substituents. The relative adsorptivity of the compounds tested was: urea < fenuron < methylurea < phenylurea < monuron < monolinuron < diuron ≤ linuron < neburon < chloroxuron (1965a, 1969b). The cation exchange capacity, pH, and clay content of the soils were not well correlated with the degree of adsorption (1965a, 1969a). Adsorption of diuron on hydrophilic soil constituents was less than on the organic fraction of soil (1965b). Linuron adsorption was determined partly by the size of the soil aggregates (Grover and Hance 1970).

There was no significant correlation between degradation and adsorption. Degradation decreased as soil organic matter was extracted, with some suggestion that the decreased degradation was due to a less favorable environment for microorganisms (Hance 1974). On alluvial clay containing 1.7 percent organic matter, diuron was more rapidly lost from soil if soil was irrigated one week after treatment rather than immediately afterwards (Horowitz and Herzlinger 1974).

Between soil and water--Monuron treatment of soil at agricultural levels led to groundwater contamination in wet years or if monuron was applied in spring, but the levels of contamination were not specified (Leh 1968). In an aquatic environment, monuron levels were higher in water than in soil for 32 weeks after treatment, then soil levels were higher than water levels (Frank 1966). Granules of monuron were phytotoxic ten meters from the point of application if transport was through water; soil transport was characterized as limited and levels of application were not available (Bersonova 1968). Neither linuron nor diuron was removed from water by slow sand filtration (Bauer 1972).

Into organisms--All the substituted urea herbicides are absorbed by plants, where they interrupt photosynthesis (Martin 1968). Residues of monolinuron have been found in asparagus at harvest (Boerner 1966) and linuron residues were present in carrot and parsnip leaves (Del Rosario and Putnam 1973). Carrots treated with linuron contained linuron, 3-(3,4-dichlorophenyl-1-methylurea; 3,4-dichlorophenylurea, and 3,4-dichloroaniline after 117 days. Metabolites accounted for no more than 13 percent of the residues (Lokke 1974). *Aurantiaceae* treated with ten, 13 or 16 kg/ha monuron or diuron contained traces of the herbicides in unripe fruit (Khubutiya and Gigineishvili 1971). One year after the last treatment of the soil cotton seeds from diuron-treated fields contained no residues (Dalton *et al*. 1966).

Hens which were fed 0.5 ppm linuron for up to six weeks did not transmit detectable levels to the yolks of their eggs (Foster *et al*. 1972). Rats which were fed monuron at 175 mg/kg/day for 60 days accumulated unspecified amounts of the herbicide in their lungs, heart, liver, brain, kidneys and milk (in decreasing order of accumulation) and also in their bone marrow and thyroid (Fridman 1968).

Volatilization--Linuron was 80 percent volatilized from metal planchets within 48 hours but even small amounts of plant material sharply decreased its volatility (Walker 1972). In Israeli soil in July, surface-applied diuron dissipated most rapidly from dry soil, but was also lost from wet soil. Irrigation immediately after herbicide application resulted in lower losses than irrigation one week later (Horowitz and Herzlinger 1974).

Persistence--

The persistence of monuron and diuron in soils is shown in Table 18, and that of linuron and monolinuron in Table 19. In addition to these quantifiable data, numerous reports of carryover between growing seasons are found in the literature. Linuron at 2.24 kg/ha persisted to the next growing season in sandy loam in Newfoundland (Morris and Penney 1971) and in Saskatchewan (Smith and Emmond 1975); monuron at ten lbs/A (11.2 kg/ha) was phytotoxic in the third growing season after application to silty clay loam in Nebraska (Burnside *et al*. 1963, 1965) and carryover with diuron is considered common (Bryant and Andrews 1967, Upchurch *et al*. 1969). As with most pesticides, persistence increases with increasing levels of application and varies with soil and climate.

Spiridonov and Kamenskii (1971) found that soil types did not affect the rate of degradation of monuron when comparisons were made between krasnozem, meadow, chestnut, and soddy-podzolic soils, while high temperature and high rainfall decreases persistence. Degradation of substituted urea herbicides was, however, more rapid in red soil than in meadow-swampy soil under subtropical conditions (Spiridonov and Yakovlev 1967). Moyer *et al*. (1972) found that the initial concentration of linuron affected its rate of degradation while clay increased its persistence only slightly, and charcoal had no significant effect. In

Table 18. PERSISTENCE OF MONURON AND DIURON IN SOILS

	% residue	Mean # days	% loss/day	Conditions	Reference
Monuron	0	1825	0.05	67 kg/ha	Isensee et al. 1973
	0	>1095	<0.09	84-112 kg/ha, S.W. USA	Ibid.
	0	>1095	<0.29	silt loam soil	Erickson 1965
	0	> 480	<0.20	Nebraska silty clay loam	Burnside et al. 1963
	0	365	0.27	0.5 ppm on sandy loam	Shadbolt et al. 1964
	0.0005	365	0.27	4.5 ppm on sandy loam	Ibid.
	0	< 365	>0.27	84-112 kg/ha, Tenn.	Isensee et al. 1973
	0	150	0.67	meadow-bog, bioassay	Spiridonov et al. 1972
	0	>60	<1.67	southern Turkmenistan	Akimov 1966
	0	60	1.67	Italy, 1.5,3.0 kg/ha	Quaglia 1960
Diuron	0	>3110	<0.03	massive levels	Isensee et al. 1973
	0	>900	<0.11	California ag. soils	Lange et al. 1968
	0	>365	<0.27	microbially poor	Ismailov and Bersunova 1968
	0	322	0.31	5 x ag. levels	Bryant and Andrews 1967
	0	112	0.89	Puerto Rican soil	Liu 1974
	0	110	0.91	meadow-bog, bioassay	Spiridonov et al. 1972
	0	105	0.95	1.5 lb/A, bioassay	Harris and Sheets 1965
	0	50	2.00		Kostowska et al. 1971
	0	>22	<4.55	ag. levels	Schweizer and Holstun 1966

Table 19. PERSISTENCE OF LINURON AND MONOLINURON IN SOILS

	% residue	Mean # days	% loss/day	Conditions	Reference
Linuron	0	>365	<0.27	>5 kg/ha light soils	Zurawski and Ploszynski 1970
	30	365	0.19	Saskatchewan soils	Smith and Emmond 1975
	0	<365	>0.27	1.5 kg/ha, light soils	Zurawski and Ploszynski 1970
	0.4	300	0.33	soil	Boerner 1965
	25	90	0.83	soil	Ibid.
	40	30	2.00	soil	Ibid.
	40	30	2.00	≤2 kg/ha, sandy soil	Homburg and Smit 1964
Monolinuron	0.2	300	0.33	soil	Boerner 1965
	5.6	90	1.05	soil	Ibid.
	0	<90	>1.11	≤2 kg/ha, sandy soil	Homburg and Smit 1964
	20	60	1.33	sandy loam soil	Schuphan 1974a
	16.4	30	2.79	soil	Boerner 1965
	60	30	2.00	≤2 kg/ha, sandy soil	Homburg and Smit 1964

comparisons among 11 soils, linuron decomposed more rapidly in soils with a high level of organic matter, primarily due to the greater microbial activity (Dubey *et al.* 1966). Coarse soils led to greater persistence of linuron than fine soils (Burnside *et al.* 1969).

Higher temperature and higher humidity or soil moisture decrease persistence of substituted urea herbicides (Burnside *et al.* 1969). When 87 to 113 kg/ha were applied to soils in the southwestern U.S., weed control persisted for over three years, but in Tennessee 84 to 113 kg/ha provided control for less than one year; soil differences as well as climate may have contributed (Isensee *et al.* 1973).

In Puerto Rico, monuron and diuron were more persistent than prometryne (Liu *et al.* 1970a, 1971) and diuron was more persistent than bromacil or fenac in a Puerto Rican forest soil, but less persistent than prometone or picloram (Dowler *et al.* 1968). Horowitz (1969) found diuron to be more persistent than atrazine, fluometron, trifluralin and prometryne in the field and greenhouse. Bromacil, noruron, ametryne, and pyrazon were also less persistent in the greenhouse but were not tested in the field. Simazine was as persistent as diuron, but no herbicide was more persistent than diuron (Horowitz 1969). Diuron was also more persistent than monuron under greenhouse conditions (Arle *et al.* 1965), and more persistent than linuron in sandy loam soils (Upchurch *et al.* 1969). Linuron also decomposed faster than monolinuron (Homburg and Smit 1964).

One year after the last of six annual applications of 2.4 lbs/A (2.7 kg/ha) of monuron, residues were 1.6 lbs/A (1.8 kg/ha) in a silt loam soil. For diuron, residues were 2.8 lbs/A (3.1 kg/ha) one year after the sixth annual application of 2.4 lbs/A (2.7 kg/ha) and 10.6 lbs/A one year after the sixth annual application of 7.2 lbs/A (8.1 kg/ha) (Dawson *et al.* 1968).

in river water which was exposed to natural and artificial light in jars, 20 percent of the monuron persisted for four weeks, and none for eight weeks (Eichelberger and Lichtenberg 1971).

Effects on Non-Target Species

Microorganisms--As photosynthetic inhibitors, the substituted urea herbicides are markedly toxic to algae. Pillay and Tchan (1972) ranged the toxicity of various herbicides to *Chlorella* and *Scenedesmus* as: diuron > neburon > monuron > atrazine > simazine > atratone.

When technical diuron was incubated with soil at levels equalling 11,227 kg/ha (five tons/A), carbon dioxide evolution was inhibited over a 56 day period. The inhibition was even more marked when formulated diuron (wettable powder) was incubated. Bacteria and fungi were inhibited by technical diuron, and bacteria were also inhibited for formulated diuron. Streptomycetes were stimulated by formulated diuron but inhibited by technical diuron (Stojanovic *et al.* 1972a). Diuron at 0.5 mM (116 ppm) induced mutations in *Chlorella* (Kvitko *et al.* 1971). Photosynthesis in *Chlorella pyrenoidosa* was inhibited by 10^{-7}M of diuron (0.023 ppm); 10^{-4}M (23.3 ppm) inhibited thermogenesis in light, and 10^{-5}M (2.33 ppm) inhibited all energy storage processes (Petrov *et al.* 1974). Diuron also induced mutants which were resistant to its inhibition of phosphorylation in *Chlorella pyrenoidosa* (Mukhamad'ev *et al.* 1971).

Zooplankton was inhibited by monuron applied to reservoirs against *Microcystis* but oxygen depletion was considered the primary cause of reductions of 35 percent, lasting up to two years (Pidgaiko and Shcherban 1970). At two ppm monuron depressed reproduction of planktonic cladocera by 50 percent (Shcherban 1971).

Among bacteria, *Azotobacter chroococcum* was more resistant to monuron and diuron than was *A. galophilum* (Babak 1968). In liquid cultures, the production of free amino acids by microorganisms was altered by linuron at levels of five ppm (Balicka et al. 1970). The inhibition of bacteria and fungi by linuron lasted for more than 145 days in sand or compost, but increases in both fungi and bacteria were observed (Lode 1967). Beck (1970) reported increases of bacteria and of CO_2 production for less than one month after monolinuron treatment; nitrification remained somewhat inhibited for 3.5 months, albeit not significantly. Fungi can to some extent be controlled by substituted urea herbicides (Goguadze 1966, Ebner 1965) and fungicidal levels of linuron are mutagenic in *Rhizobium* (Kaszubiak 1968).

The effects of monuron, linuron, diuron and monolinuron on soil processes are shown in Tables 20 and 21. The effects of diuron on soil microorganisms and aquatic microorganisms are shown in Tables 22 and 23, respectively.

At agricultural levels, linuron did not affect ammonifying bacteria (Rankov et al. 1967) or oligonitrophilic bacteria (Ulasevich et al. 1973) but inhibited nitrifying bacteria (Rankov et al. 1966, Torstensson 1974). High levels of linuron also inhibited ammonifying and cellulolytic bacteria (Torstensson 1974, Rankov et al. 1967). Overall numbers of soil bacteria were unaffected by four kg/ha of linuron while eight kg/ha in chernozem inhibited bacteria below ten cm, but stimulated their numbers above ten cm (Ulasevich et al. 1970, 1973).

When specific genera were tested, linuron almost invariably depressed growth. Only *Pseudomonas fluorescens* at 2.5 to 4.0 ppm (Sobieszczanski 1969) and *Pseudomonas phaseoli* (Balicka and Krezel 1969) were not affected. *Azotobacter* was strongly inhibited by linuron (Ulasevich et al. 1973, Wegrzyn 1971, Pajewska 1969) with levels as low as 1.5 ppm. *Bacillus cereus* and *Rhizobium meliloti* were inhibited by 500 to 1,000 ppm in the laboratory and under field conditions (Pajewska 1969) and 2.5 to 4.0 ppm were toxic to *Arthrobacter*, *Corynebacterium* and *Bacillus* species in culture (Kosinkiewicz 1973, Sobieszczanski 1969).

Linuron was toxic to all algae tested, *Chlorella* being inhibited at 0.2

Table 23. EFFECTS OF DIURON ON SOIL PROCESSES

Parameter	Effect	Level Original	 PPM	# days observed	Conditions	Reference
Nitrification	0	5 ug/g	5			Corke and Thompson 1970
	-	100 ug/g	100		3 day inhibition	Ibid.
	-	20 ppm.	.3		low-nitrifying soil	Dubey 1969
	0	20 ppm	20		highly nitrifying soil	Ibid.
	0	1-4 mg/kg	1-4		2 soils, Brazil	Kuramoto et al. 1970
	0	<10 lbs/A	<5			Caseley and Luckwill 1964
	+	12 kg/ha	5.4		in soil	Zavarzin and Belyaeva 1966
	+	1.5 kg/ha	0.68		meadow soil	Tulabaev and Tamikaev 1968
	0	8 kg/ha	3.6		Armenian vineyard	Akopyan and Agaronyan 1968
Respiration	+	10 ppm	10		loam, clays	Liu and Cibes-Viade 1972
	-	100 ppm	100		loam, clays	Ibid.
	0			<60	composting	Zinchenko and Osinskaya 1969
	+			>60	composting	Ibid.
	0	ag levels				Corke and Robinson 1960
Ammonification	+	6 kg/ha	2.7		orchard soil	Arshidinov et al. 1974
Urease	+				composting	Zinchenko and Osinskaya 1969
Catalase	+				composting	Ibid.
P_2O_5 available	-	12 kg/ha	5.4	365	in soil	Zavarzin and Belyaeva 1966
K available	-	6 kg/ha	2.7		orchard soil	Arshidinov et al. 1974

+ = stimulation   0 = no effect   - = inhibition

Table 11. EFFECTS OF LINURON, MONURON, AND MONOLINURON ON SOIL PROCESSES

Parameter	Effect	Level Original	Level PPM	Conditions	Reference
Linuron:					
Respiration	–	500 ppm	500	laboratory	Grossbard 1971
	+	$\leq 4 \times 10^{-5}$ M	≤10	maize rhizosphere	Karpiak and Iwanowski 1969
	0	10 ppm	10	clay soil	Liu and Cibes-Viade 1972
	–	100 ppm	100	clay soil	Ibid.
Nitrification	0	5 µg/g	5	soil	Corke and Thompson 1970
	–	100 µg/g	100	soil	Ibid.
	–	2%	20,000	in vitro	Rankov 1966a
Monolinuron:					
Respiration	0	ag levels			Balicka and Sobieszczanski 1969
Ammonification	+			soil	Peshakov *et al.* 1969
Nitrification	–			soil	Ibid.
Monuron:					
Respiration	0	ag levels		Warburg apparatus	Kulinska 1967
	0	ag levels			Corke and Robinson 1960
Ammonification	+	4-12 kg/ha			Zavarzin 1966
Nitrification	+	4 kg/ha	8.8	soil	Zavarzin and Belyaeva 1966
	–	4-12 kg/ha	8.8-13.2		Zavarzin 1966
	–	1.5 kg/ha	3.30		Tulabaev 1971
	0	8 kg/ha	17.2	Armenian vineyard	Akopyan and Agaronyan 1968

+ = stimulation 0 = no effect – = inhibition

Table 22. EFFECTS OF DIURON ON SOIL MICROORGANISMS

Parameter	Effect	Level Original	 PPM	Conditions	Reference
Soil microflora	0	4-6 kg/ha	1.8-2.7	orchard soil, chernozem	Arshidinov et al. 1974
	+	1.5 ppm	1.5	pots	Sharma and Saxena 1970
Bacteria	–	10%	100,000	mud, Turkmen SSR	Gel'tser et al. 1972
Cellulolytic	0	1.5 kg/ha	0.7	meadow soil	Tulabaev and Tamikaev 1968
	+	8 kg/ha	3.6	Armenian vineyard	Akopyan and Agaronyan 1968
Ammonifying	–	1.5 kg/ha	0.7	meadow soil	Tulabaev and Tamikaev 1968
Nitrifying	–	8 kg/ha	3.6	Armenian vineyard	Akopyan and Agaronyan 1968
Azotobacter chroococcum	–	3-7.5 ppm	3-7.5	pots	Sharma and Saxena 1970
Arthrobacter	–	0.05 mg/l	0.05	laboratory	Gogoadze 1968
	–	0.5%	5,000	mud, Turkmen SSR	Gel'tser et al. 1972
	–	8 kg/ha	3.6	Armenian vineyard	Akopyan and Agaronyan 1968
Pseudomonas	–	0.5%	5,000	mud, Turkmen SR	Gel'tser et al. 1972
P. fluorescens	–	25 ug/l	0.025		Breazeale and Camper 1972
Erwinia carotovora	–	25 ug/l	0.025		Ibid.
Bacillus sp.	–	25 ug/l	0.025		Ibid.
Fungi	+	3 ppm	3	pots	Sharma and Saxena 1970
	0	1.5 kg/ha	0.7		Tulabaev 1971
Actinomycetes	0	3-7.5 ppm	3-7.5	pots	Sharma and Saxena 1970

+ = stimulation 0 = no effect – = inhibition

Table 23. EFFECTS OF DIURON ON AQUATIC MICROORGANISMS

Parameter	Effect	Level Original	 PPM	# days observed	Conditions	Reference
Oxygen evolution:						
Chlorophyceae	-	22 ppb	0.022			Hollister and Walsh 1973
Bacillariophyceae	-	67 ppb	0.067			Ibid.
Chrysophyceae	-	13 ppb	0.013			Ibid.
Rhodophyceae	-	24 ppb	0.024			Ibid.
Photosynthesis:						
Chlorella pyrenoidosa	-	10^{-7} M	0.023		55% reduction	Petrov *et al.* 1974
	-	10^{-6} M	0.233		total inhibition	Ibid.
Nitrogen fixation:						
Anabaena sp.	-	≥ 10 ppm	≥ 10			Venkataraman and Rajyalakshmi 1972
Nostoc sp.	-	≥ 5 ppm	≥ 5			Ibid.
Anacystis nidulans		2000 ppm	2000			Ibid.

+ = stimulation   0 = no effect   - = inhibition

Table 13. Cont'd

Parameter	Effect	Level Original	Level PPM	# days observed	Conditions	Reference
Growth:						
Chlorella sp.	-	0.0004 ppm	.0004	>14		Ukeles 1962
Chlorella pyrenoidosa	-	0.1 ppm	0.1	>1	90% inhibition	Virmani et al. 1975
	-	2 ppm	2	≤1	lethal	Ibid.
Chlorococcum hypnosporum	-	0.5 ppm	0.5	>1	90% inhibition	Ibid.
	-	2 ppm	2	≤1	lethal	Ibid.
Monochrysis lutheri	-	0.00002 ppm	0.00002	14	lethal	Ukeles 1962
Phaeodactylum tricornutum	-	0.00002 ppm	0.00002	14	17% inhibition	Ibid.
Dunaliella euchlora	-	0.00002 ppm	0.00002	14	20% inhibition	Ibid.
Protococcus sp.	-	0.00002 ppm	0.00002	14	48% inhibition	Ibid.
Scenedesmus quadricauda	-	0.1 ppm	0.1		>95% inhibition	Stadnyk et al. 1971
Zooplankton:						
Scapholeberis mucronata	-					Pidgaiko 1971

+ = stimulation 0 = no effect - = inhibition

to 1.0 ppm (Knauf and Schulze 1972). For *Anabaena* species, inhibiting levels of linuron varied from one ppm to 2,300 ppm and for *Nostoc* species from 100 to 500 ppm (Venkataraman and Rajyalakshmi 1971). Da Silva et al. (1975) reported toxicity to algae from ten ppm linuron, and Pantera (1970) noted that high levels of linuron were toxic to soil algae.

In contrast, fungi were not severely affected (Peshakov et al. 1969). Four kg/ha neither stimulated nor depressed actinomycetes (Ulasevich et al. 1970, 1973) and Bakalivanov (1972) stimulated growth of *Penicillium* with linuron; eight kg/ha inhibited this fungus in chernozem, however (Ulasevich et al. 1970, 1973).

When monuron was applied to a weed-free soil at eight to 16 kg/ha, the available potassium, nitrogen and phosphorus levels of the soil were not changed (Spiridonov et al. 1972) but shifts in phosphorus levels due to similar levels of monuron have been reported (Zavarzin 1966, Zavarzin and Belyaeva 1966). Monuron stimulated soil catalase and peroxidase (Spiridonov et al. 1972).

Agricultural levels of monuron did not affect the overall numbers of soil microorganisms (Kulinska 1967a) but high levels reduced the microbial population (Pantera 1972). Effects shifted with time: Tsilynchuk (1974) noted that six to eight kg/ha of monuron reduced the microbial population of southern chernozem for thirty days, after which microbial levels in treated soils rose above those of control soils. Cellulolytic bacteria in a bog soil increased above control levels from 40 to 70 days after the soil was treated with eight to 16 kg/ha of monuron, but fungal numbers decreased (Spiridonov et al. 1972). In a meadow soil, 1.5 kg/ha decreased the numbers of nitrifying and ammonifying bacteria, but not of cellulolytic bacteria (Tulabaev 1971, Tulabaev and Tamikaev 1968). Nitrifying bacteria were also inhibited by eight kg/ha in an Armenian vineyard (Akopyan and Agaronyan 1968).

Monuron inhibited algal growth (Mikhailova and Kruglov 1973): Soil algae were stimulated by 0.001 to 0.01 ppm, but 0.1 to 1.0 ppm was toxic

(Pillay and Tchan 1972). Nitrogen fixation by *Chlorogloea* was inhibited by 10 ppm, but 25 ppm were required to stunt growth (DaSilva et al. 1975). Ukeles (1962) observed inhibition of five species of phytoplankton at 0.02 ppm, and one species (*Phaeodactylum tricornutum*) was inhibited by 0.01 ppm.

Monolinuron did not alter the numbers of fungi or actinomycetes in soil (Peshakov et al. 1969) nor the numbers of anaerobic bacteria (Sobotka 1970) but did inhibit the growth of nitrifying, denitrifying and ammonifying bacteria (Sobotka 1970). The fungus *Suillus variegatus* was not affected by four kg/ha (Sobotka 1970). *Azotobacter chroococcum* was inhibited by 100 or 1,000 ppm but not by one or ten ppm (Wegrzyn 1971) but *Chlorella* was sensitive to 0.2 ppm monolinuron during a four day incubation (Knauf and Schulze 1972).

Although effects on microorganisms other than algae range from stimulating to inhibiting, the predominance of inhibitory effects makes it apparent that these substituted urea herbicides do alter the numbers and kinds of soil microorganisms. Moreover, Corke and Thompson (1970) observed that, whereas 100 ppm of linuron or diuron are required to inhibit nitrification for three days, 2.5 to 5.0 ppm of their metabolite, 3,4-dichloroaniline, is very toxic to *Nitrosomonas*, and 25 ppm of the metabolite 3-(3,4-dichlorophenyl)-1-methylurea strongly inhibit nitrification.

Invertebrates--The LC$_{100}$ of linuron to *Daphnia magna* was six ppm, but to *Artemia salina*, greater than 100 ppm. Snails (*Lymnaea stagnalis*) and aquatic worms (*Tubifex*) were not injured by 0.2 to one ppm (Knauf and Schulze 1972). Shcherban (1972) observed adaptation of some *Cladocera* to diuron and Braginskii and co-workers (1972) noted that whereas low concentrations of diuron inhibited zooplankton, higher concentrations were stimulatory.

When clams (*Mercenaria mercenaria*) were exposed to 0.25 ppm diuron, 92 percent of the eggs developed and larval survival was not decreased; at five ppm larvae also survived, but no eggs developed. For monuron, five ppm inhibited neither the development of oyster eggs nor the survival of their larvae (Davis and Hidu 1969).

Among land invertebrates, monuron was mutagenic at 0.01 percent (100 ppm) in *Drosophila melanogaster* (Shkvar et al. 1969) and monolinuron at 0.1 mg/100g soil (one ppm) inhibited reproduction in *Folsomia candida* (Sanocka-Woloszyn and Woloszyn 1970). Monuron decreased the numbers of wireworms, earthworms, springtails, millipedes, and mites in grassland soil at levels above 11.2 kg/ha (Fox 1964).

Fish and amphibians--The 48-hour LC_{50} of diuron to coho salmon (*Oncorhynchus kisutch*) and largemouth bass (*Micropterus salmoides*) was 42 ppm and 16 ppm, respectively (Pimentel 1971). Bluegills (*Lepomis macrochirus*) exposed to 3.0 ppm of diuron in ponds died as a consequence of oxygen depletion (McCraren et al. 1969) while roach fish (*Rutilus rutilus*) developed methemoglobinemia as a consequence of diuron poisoning (Komarovskii 1973). Popova (1970) reported that diuron at 0.2 mg/liter (0.2 ppm) decreased piscine hemoglobin content and the number of erythrocytes within five days. Both monuron and diuron accumulated in roach fish exposed to 0.2 to 0.3 mg/liter (0.2-0.3 ppm) for two to three months (Komarovskii and Popovich 1971).

The 24-hour LC_{50} of monuron for the channel catfish (*Ictalurus punctatus*) was 75 ppm and the 48-hour LC_{50} for the mullet (*Mugil cephalus*) was 16.3 ppm (Pimentel 1971). Ten ppm of monuron were toxic to some freshwater fish within eight days (Pimentel 1971).

Birds--When two-week old birds were fed diuron for five days followed by clean feed for three days, 14 percent of quail, 33 percent of pheasants, and 30 percent of mallards died of 5,000 ppm, while the LC_{50} for bobwhites was 1,730 ppm with 95 percent confidence limits between 1,482 ppm and 2,035 ppm. No bobwhite died when fed 5,000 ppm monuron, but 21 percent of the quail and ten percent of the mallards died, while the LC_{50} for pheasants was calculated to be 4,682 ppm, with 95 percent confidence limits of 3,902 to 5,746 ppm (Heath *et al*. 1972). When linuron or diuron was injected into fertile hens' eggs, 100 ppm of the former or 200 ppm of the latter decreased the hatching rate significantly (Dunachie and Fletcher 1967, 1970).

Mammals--The acute oral LD_{50} of diuron is 3,400 mg/kg; of linuron 4,000 mg/kg; of monuron, 3,500 to 3,700 mg/kg; and of monolinuron, 2,250 mg/kg (Martin 1969, Pimentel 1971).

Monuron is carcinogenic if rats are fed 450 mg/kg (450 ppm) per day for 17 months (Rubenchik *et al*. 1969, 1970) or if mice are fed six mg/week for 15 weeks (Rubenchik *et al*. 1970). Diuron was less strongly carcinogenic, with fewer carcinomas occurring after a longer latency period (Rubenchik *et al*. 1973). Rosival (1970) characterized urea herbicides as indirect mutagens, perhaps because of their demonstrated cytogenic effects (Khubutiya and Ugulava 1973), and their mutagenicity in microorganisms and in plants (Wuu and Grant 1966).

Dystrophic changes in rat livers due to linuron and diuron have been reported after chronic feeding of ten percent of the acute LD_{50} (Bankowska and Bojanowska 1973) and monolinuron at 230 to 460 mg/kg/day caused damage to the liver, kidneys and spleen of rats (Pasiewicz and Nikodemska 1973). Air levels of 13 mg/m^3 reportedly caused changes in the erythrocyte count of rats (Lomonova 1969).

Conclusions-

Despite the wealth of data on the effects of the herbicides monuron, linuron, diuron and monolinuron on soil processes and soil organisms, the consequences of disposing of these compounds in soil are unclear. Their extreme toxicity to algae, considerable toxicity to bacteria and fungi, and their relatively great mobility and persistence argues against soil disposal. Bulk disposal would invite pollution of [illegible] soil and water for years. Disposal of small amounts over larger areas would probably be acceptable at normal agricultural use levels.

EPTC

EPTC, the common name for *S*-ethyl di-*n*-propyl thiocarbamate, was introduced by Stauffer Chemical Co. in 1955 under the name Eptam or Eradicane as a selective preemergence herbicide. EPTC is a light yellow colored liquid with an amine odor, a boiling point of 127°C at 20 mm mercury, a vapor pressure of 0.1 mm mercury at 24°C and a flash point of 116°C. Its water solubility is 370 ppm at 20°C and it is completely miscible with acetone, ethanol, and kerosene at 20°C. EPTC is synthesized by the reaction of ethyl mercaptan and di-*n*-propylcarbamoyl chloride in the presence of base, or by the reaction of di-*n*-propylamine with *S*-ethyl chloroformate.

Degradation-

In one study the degradation of EPTC proceeded at similar rates in sterilized and unsterilized soil, and after seven weeks in this heavy clay soil phytotoxicity was lost although very little carbon dioxide was evolved (MacRae and Alexander 1965). Other studies contradicted these results. In five soil types including peaty muck and pure sand inactivation decreased sharply when soil was sterilized (Koren et al. 1968). Sheets (1959) estimated that degradation in sterilized soil was one third that of non-autoclaved soil. More rapid degradation occurred in loam than in heavy clay soils (Smith and Fitzpatrick 1970), and when soil moisture was increased (Cialone and Sweet 1963, Fang 1969). Fang (1969) suggested that the low levels of $^{14}CO_2$ evolution could be due to microbial utilization of the ethyl moiety of EPTC. No microorganisms capable of degrading EPTC have been identified, nor were data available on microorganisms which failed to decompose EPTC (Fang 1969, Menzie 1974).

Adult female rats fed 0.6 to 103 mg EPTC excreted $^{14}CO_2$ and six urinary metabolites, including urea (Menzie 1974). In mice, degradation proceeded by sulfoxidation followed by cleavage of the thiocarbamate ester group. EPTC-sulfone was six percent degraded to carbon dioxide, but EPTC and its sulfoxide were 35 to 41 percent converted to carbon dioxide (Casida *et al.* 1975).

Chemical and physical--EPTC is decomposed by concentrated acids at high temperatures, but treatment with 10*N* sodium hydroxide for one hour at 95°C had little effect (Smith and Fitzpatrick 1970).

Transport--

Within soil--EPTC was more readily adsorbed to dry soil than wet (Leuchenko and Gortlevskii 1970, Koren and Ashton 1969) and more readily at low temperatures than at high (Koren and Ashton 1969). Complexes of EPTC with montmorillonite were stable against some moisture increases, but could be reversed by complete water saturation (Mortland and Meggitt 1966). On model aliphatic adsorbents, EPTC adsorption increased with increasing length of the alkyl group from C_8 to C_{18}, and more EPTC adsorbed to the peat fraction of soil than to the humic acid fraction (Hance 1969b).

In soil columns, EPTC leaching increased as temperatures dropped from 24° to 3°C (Vernetti and Freed 1963). Leaching was not significant in dissipating unspecified levels of EPTC from a black clay loam over a 90 day period (Yatsenko and Baikova 1971). In a peaty muck, EPTC leached less than five cm (Koren *et al.* 1969). No data were available on the transport of EPTC from soil into water or from water into sediments.

Volatilization--When EPTC was applied to wet soil, 22 to 38 percent volatilized within one hour and volatilization from dry soil was even greater (Gray 1965). Its volatilization from peaty muck, adobe clay, clay loam and sandy loam was 15.1 percent, 16.4 percent, 5.3 percent, 10.6 percent and 18.1 percent, respectively, within one hour and the greatest amount of volatilization was from pure sand (Koren *et al.* 1968, 1969).

Persistence--

The phytotoxicity of agricultural levels of EPTC persists for one week in moist loam at 70° to 80°F (WSSA 1974) and for not more than 90 days in black clay loam planted to sugar beets (Yatsenko and Komissarov 1970). Persistence was estimated to be less than one growing season when four to six kg/ha EPTC were applied in sugar beet fields, while eight kg/ha applied before sowing often resulted in carryover to the next season (Yatsenko and Blakova 1971). Four ppm (8.96 kg/ha) of EPTC retained its phytotoxicity for three months and persistence was correlated with the organic matter, clay, and cation exchange capacity of the soil (Sheets 1959). Wet clay granules were less persistent than dry clay granules (Cialone and Sweet 1963).

Effects on Non-Target Species--

Microorganisms--At agricultural levels, EPTC did not alter the overall numbers of microorganisms in the soil (Balicka and Sobieszczanski 1969, Zharasov and Chulakov 1972, Lobanov and Poddubnaya 1968). Nitrifying bacteria were inhibited on dark chestnut soil (Zharasov 1972) and on gray podzol or chernozem (Nikolaenko 1970, Nikolaenko et al. 1970), but stimulated after two months at an unspecified level (Novogrudskaya et al. 1965). In cotton fields, eight kg/ha had no effect on nitrifying bacteria (Tulabaev 1970) and *Azotobacter* cultures were not inhibited by one or ten ppm but were inhibited by 100 or 1000 ppm (Wegrzyn 1971).

Different types of bacteria were variably affected by EPTC with cellulolytic bacteria inhibited by levels of eight kg/ha or higher (Sobieszczanski 1969, Tulabaev 1970) and saprophytic bacteria in leached chernozem stimulated at the same level (Matenova and Semikhatova 1973). Denitrifying bacteria in cotton fields were stimulated by eight kg/ha (Tulabaev 1970) or on agar after 1.5 months exposure to EPTC (Novogrudskaya et al. 1965) but the same bacteria were inhibited on gray podzol or chernozem (Nikolaenko 1970). Ammonifying bacteria in cotton fields were unaffected by eight kg/ha (Tulabaev 1970) but inhibited on gray podzol or chernozem (Nikolaenko 1970).

When different species were tested for sensitivity to EPTC, bacilli were most resistant, with *Bacillus megaterium*, *B. mesentericus* and *B. vulgatus* being stimulated by unspecified levels in leached chernozem (Mataneva and Semikhatova 1973) and *B. subtilis* being unaffected by 1000 ppm in paper-disc culture (Thomas et al. 1973). *Azotobacter* was stimulated by EPTC on leached chernozem (Mataneva and Semikhatova 1973), unaffected by eight kg/ha in cotton fields (Tulabaev 1970) and by one ppm in culture, but inhibited by 100 ppm in culture (Wegrzyn 1971).

Fungi were inhibited by agricultural levels of EPTC as well as by 20 mg/ml (20,000 ppm) in light and dark chestnut soils (Zharasov 1972, Zharasov et al. 1972) but not on leached chernozem (level unspecified) (Mataneva and Semikhatova 1973). Fungal species inhibited by 4.5 kg/ha (10 ppm) were *Cladosporium*, *Alternaria*, and *Curvularia* (Zharasov et al. 1972) while sclerotial growth, but not fungal numbers, of *Sclerotium rolfsii* was inhibited by 20 ug/g (20 ppm) EPTC (Rodriguez-Kabana and Curl 1972). Soil algae were reportedly unaffected by EPTC, but levels were not specified (Mikhailova and Kruglov 1973). In aquaria, 0.5 to 1.0 mg/ml (500 to 1000 ppm) depressed the carbon dioxide evolution and increased the ammonia levels of the water; planktonic growth was not affected (Pischolka 1971).

The effects of EPTC on soil processes varied. Nikolaenko (1970) reported that ammonification was inhibited by EPTC in gray podzol or chernozem. No effects on nitrification were observed when 4.5 kg/ha were applied to sugar beet fields of light chestnut soil (Zharasov 1971, Zharasov and Chulakov 1972) and unspecified amounts of EPTC stimulated nitrification on sandy-loamy chernozem (Lobanov and Poddubnaya 1968). Cell suspensions of *Nitrobacter* were inhibited by ten ug/ml (10 ppm) (Winely and San Clemente 1970). Soil respiration was reportedly enhanced by EPTC (Karpiak and Iwanowski 1969) or unaffected (Balicka and Sobieszczanski 1969). The data suggest that EPTC is somewhat fungicidal and affects bacteria selectively. The toxicity of EPTC to microorganisms was greater on gray podzolic soil than on chernozem, although its phytotoxicity was greater on chernozem (Nikolaenko 1970). Heavy low-humus chernozem resulted in lower toxicity of EPTC to nitrogen-fixing bacteria than did solonized chernozem (Nikolaenko et al. 1970). As the moisture content of soil fell, microbial toxicity increased (Nikolaenko and Geller 1969). EPTC uncoupled oxidative phosphorylation in *Nitrobacter agilis* to some extent (Vinely and San Clemente 1971).

Invertebrates--EPTC was not considered toxic to honey bees by Morton and co-workers (1972) but did inhibit development of ova and of larvae at 100 ppmw (Morton and Moffett 1972). The LC_{50} of EPTC for *Daphnia magna* was 4.7 ppm at 48 hours and 9.4 ppm at 96 hours (Koval'chuk et al. 1971). Fifty percent of white shrimp (*Penaeus setiferus*) were paralyzed or killed by exposure to 0.63 ppm for 96 hours, and shell growth was inhibited in eastern oysters (*Crassostrea virginica*) exposed to five ppm for 96 hours (Pimentel 1971).

Vertebrates--White mullet (*Mugil curema*) had an LC_{10} of 20 ppm EPTC in 48 hours (Pimentel 1971) and the 96-hour LC_{50} was 17 ppm in mosquito fish (*Gambusia affinis*) (WSSA 1974).

In bobwhite quail (*Colinus virginianus*) the oral LD_{50} is 20,000 ppm per day for seven days (WSSA 1974) and the acute LD_{50} for chick embryos is 0.74 mg/kg (Medved et al. 1970). No teratogenesis was seen in rats fed 0.5 or 5.0 percent of the LD_{50} of EPTC throughout pregnancy but the higher level was highly embryotoxic (Medved et al. 1970). Olefir (1973) reported that rats fed the maximum tolerated level of EPTC for one week exhibited decreased immune capacity. The acute oral LD_{50} of EPTC is 1,630 mg/kg for mice and 3,160 mg/kg for rats (Pimentel 1971).

Conclusions:

The disposal of EPTC in soil is feasible, since this herbicide is neither overly persistent nor highly toxic to soil organisms. Some fungicidal activity as well as changes in the numbers and species of soil microorganisms would almost certainly occur, and some leaching must be anticipated. The degree of water pollution to be anticipated cannot be determined from the data.

Nitralin and Trifluralin

Nitralin is the common name for 4-(methylsulfonyl)-2,6-dinitro-*N*,*N*-dipropylbenzamine, introduced by the Shell Chemical Co. in 1966 as a selective preemergence herbicide on numerous vegetables, fruits, turf and ornamentals and soybeans. It is a yellow crystalline solid with a mild odor which melts at 151^{0}C to 152^{0}C, which has a vapor pressure of 1.8×10^{8} mm mercury at 25^{0}C, and decomposes vigorously at 225^{0}C, with an estimated heat of decomposition of 250 cal/gram. Nitralin has a water solubility of 0.6 ppm , 37 percent (370,000 ppm) in acetone, and 33 percent (330,000 ppm) in dimethyl sulfoxide at 25^{0}C. It is inflammable but not corrosive.

Trifluralin is the common name for 2,6-dinitro-*N*,*N*-dipropyl-4-(trifluoromethyl) benzamine, introduced by Elanco Products Co. in 1959 as a preemergence herbicide in vegetables, fruit, nut trees and soybeans. It is an orange crystalline solid with a melting point of 48.5^{0} to 49^{0} C, a boiling point of 96^{0} to 97^{0}C at 0.18 mm mercury, and a vapor pressure of 1.99×10^{-4} mm mercury at 29.5^{0}C. Its water solubility is less than one ppm, but it is soluble in ethanol to 7 g/100 ml (70,000 ppm) and in acetone to 40 g/100 ml (400,000 ppm). Trifluralin is neither inflammable nor corrosive. Technical trifluralin is at least 95 percent pure and melts above 42^{0}C.

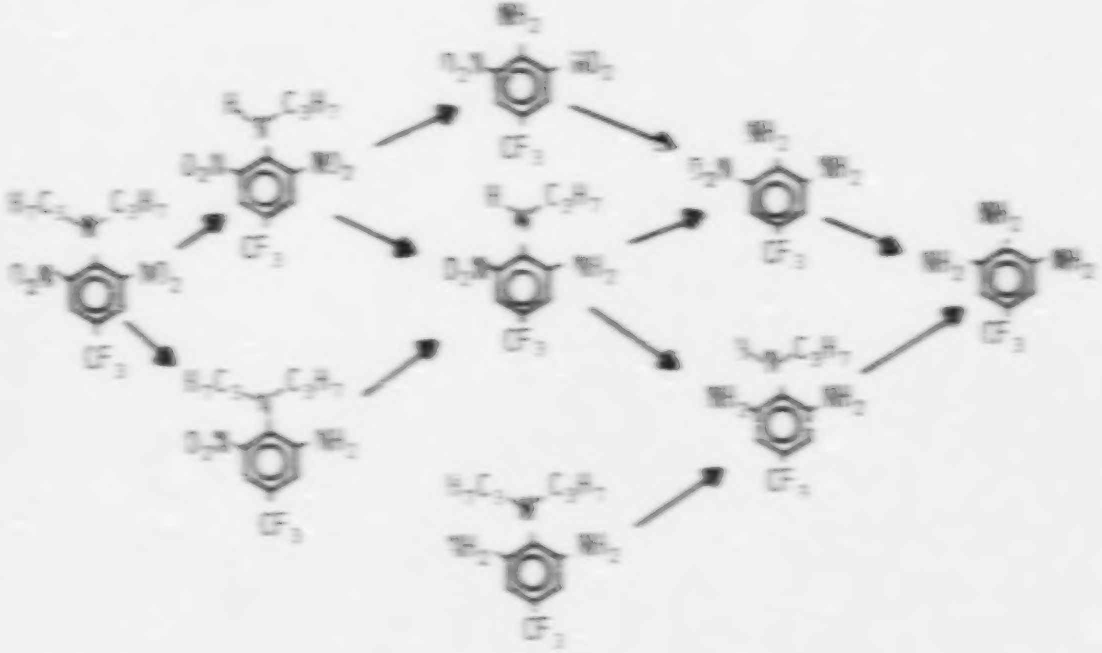
Helling (1976) has recently reviewed the behavior of dinitroaniline herbicides in soil.

Degradation-

Biological--Hamdi and Tewfik (1969) reported that pseudomonads degraded trifluralin by splitting the nitro-groups from the aromatic moiety. Patrosini et al. (1970) found significantly greater decomposition of trifluralin in unsterilized soil than in sterilized soil, with additional stimulation of degradation if organic matter was added to the soil. When $^{14}CF_3$-trifluralin was incubated with *Paecilomyces*, *Fusarium oxysporum* or *Aspergillus fumigatus*, less than one percent of the radioactivity evolved as $^{14}CO_2$ (Laanio et al. 1973), and Willis and coworkers (1974) observed little correlation between trifluralin degradation and microbial respiratory activity in soil suspensions. Degradation was enhanced by anaerobic conditions if the oxidation-reduction potential of the soil was lowered by prestimulating microbial activity (Willis et al. 1974). Nitralin degradation rates were not affected by activated carbon added to soil, but trifluralin degradation was (Talbert and Kennedy 1972).

Trifluralin decomposed more rapidly at soil moisture equal to 160 percent of field capacity than at 80 percent of field capacity in loam soils (Messersmith et al. 1971). In silt loam under laboratory conditions, trifluralin in aerobic autoclaved soil was not significantly decomposed after 20 days, but it was decomposed in unautoclaved soil. Most of the trifluralin losses were due to volatilization rather than degradation, however (Parr and Smith 1973). The products of trifluralin degradation are shown in Figure 4. Probst and Tepe (1969) concluded that, although microorganisms may contribute to destruction of trifluralin in soil, there is no evidence that microbial degradation predominates. Corbin and Upchurch (1967), investigating the effects of pH on herbicide detoxification in soil, concluded that trifluralin was not detoxified in soil. Data on the microbial degradation of nitralin are not available.

In rats and dogs fed 100 mg/kg $^{14}CF_3$-trifluralin in a single oral dose, 78 percent of the radioactivity was eliminated in the feces and 22 percent in the urine. Urinary metabolites consisted of partially and completely dealkylated compounds, but the CF_3-group remained intact. Trifluralin was poorly absorbed from the gut, resulting in high fecal excretion (Emmerson and Anderson 1966). Studies of the animal metabolism of nitralin are not available (Paulson 1975).


H7C3
C3H7
O2N
NH2
CF3
H
C3H7
O2N
NO2
CF3
NH2
O2N
NO2
CF3
H7C3
C3H7
O2N
NO2
CF3
H
C3H7
O2N
NO2
CF3
NH2
O2N
NH2
CF3
H7C3
C3H7
NH2
NH2
CF3
H
C3H7
NH2
NH2
CF3
NH2
NH2
NH2
CF3

DEGRADATION OF TRIFLURALIN IN SOIL (FROM KAUFMAN 1974)

Figure 4

Photolytic--Trifluralin on glass decomposed photolytically during four to six hours' exposure to 300-700 mµ light (Wright and Warren 1965). Photolytic decomposition proceeded by oxidative dealkylation, nitro-reduction and cyclization. The products included 2,3-dihydroxy-2-ethyl-7-nitro-1-propyl-5-trifluoromethylbenzimidazoline and 2-ethyl-7-nitro-5-trifluoromethylbenzimidazole 3-oxide, both of which could be further degraded by heat or irradiation. Under acid conditions, 2-amino-6-nitro-2,2,2-trifluoro-*p*-toluidine was formed, and under alkaline conditions, 2-ethyl-7-nitro-5-trifluoromethylbenzimidazole accounted for 80 percent of the decomposition products (Leitis and Crosby 1974).

Using a vapor-phase reactor which simulated sunlight, Soderquist and coworkers (1975) identified two dinitrotoluidines and two benzimidazoles as photolytic products of trifluralin (Figure 4). Up to 95 percent of trifluralin applied to anaerobic silt loam at 1.0 ppm and 0.1 ppm was recovered after 30 days in the dark, but only 18 percent after 30 days in the light. But little degradation of 200 ppm occurred even in light (Parr and Smith 1973). Although nitralin photodegradation may follow the same pattern as that of trifluralin, no data are available.

Chemical and physical--Trifluralin was completely decomposed by liquid ammonia with either metallic sodium or lithium, and was 91.2 percent decomposed by sodium biphenyl (Kennedy *et al*. 1972a). Burning formulated trifluralin at 900°C produced as volatile products: carbon monoxide, carbon dioxide and ammonia (Kennedy *et al*. 1972a, 1972b). Decomposition of 25 percent of trifluralin was achieved for the reference standard by heating to 879°C, and for the formulated (liquid) trifluralin at 862°C (Kennedy *et al*. 1969). Some reduction in the phytotoxicity of trifluralin was achieved by exposing 10 ppm or 100 ppm to three Mrad of gamma-irradation (Horowitz and Blumenfeld 1973).

Transport:

Within soil--Helling (1971) characterized trifluralin as immobile in all soils. Being highly volatile, trifluralin diffused in the vapor phase and its diffusion in soil was not affected by variations in soil water (Scott and Phillips 1972). Trifluralin was strongly adsorbed by activated carbon (Coffey and Warren 1969) and by various adsorbents in the order: peat moss > wheat straw ≥ cellulose triacetate > cation exchange resin > anion exchange resin > silica gel and cellulose powder > kaolinite > montmorillonite (Grover 1974). Its adsorption on muck was sufficiently great to prevent four lbs/A (4.9 kg/ha) from leaching two inches (Eshel and Warren 1967) but it could be readily desorbed from montmorillonite by water (Grover 1974). When the amount of organic matter was low and was kept constant, however, adsorption increased with increasing clay content (Horowitz et al. 1974). The level of montmorillonite clay in the soil did not affect trifluralin's bioactivity after the first year (Weber et al. 1974). Koren (1972) increased the leaching of trifluralin by adding surfactants to the soil, but characterized the degree of movement in dry soil as slight.

In silt loam in the laboratory, trifluralin diffusion increased directly with increases in bulk density to 1.1 g/cm^3, then decreased with increasing bulk density. Vapor diffusion increased about 50 percent for each ten percent increase in the air-filled porosity of the soil, and for bulk densities between 1.2 g/cm^3 and 1.4 g/cm^3, the magnitudes of vapor diffusion and solution diffusion were similar (Bode et al. 1973a). Diffusion was low in air-dry soils, increased until the soil moisture content reached eight to 15 percent (weight/weight) and then decreased again. Diffusion also decreased if the air-filled fraction of the soil fell below 40 percent (volume/volume). A model adequate to predict trifluralin diffusion in soil included 15 parameters (Bode et al. 1973b). When 0.84 and 1.68 kg/ha of trifluralin were soil incorporated, 80 percent remained in the top 15 cm, and the rest above 30 cm in fine sandy loam. After 15 months, residues in the zero to 15 cm layer were 0.11 ppm; in the 15-30 cm layer, 0.10 ppm (Miller et al. 1975). In four southern soils, trifluralin leached into the two to four inch layer (five to ten cm) in 22 weeks (Schweizer and Holstun 1966).

In contrast to trifluralin, both nitralin and its metabolites leached readily through clay loam in soil columns (Anderson *et al.* 1968). After 15 months, 0.84 to 1.7 kg/ha soil-incorporated nitralin had leached to the 15-30 cm layer (Miller *et al.* 1975).

Between soil and water--Most losses of trifluralin into surface waters occurred when heavy rains followed herbicide treatment; but, over a three year period, losses into water did not exceed 0.05 percent per season (Willis *et al.* 1975). No data were available for nitralin.

Volatilization--The phytotoxicity of surface-applied trifluralin was enhanced by adding adsorbents to the soil, presumably because the adsorbents decreased volatiliy (Bardsley *et al.* 1967). Losses due to volatilization were proportional to the concentration of trifluralin, and increased with time and with the amount of soil water when trifluralin was applied to the soil surface. Vapor loss decreased sharply when the herbicide was incorporated to a depth of 1.27 cm (Bardsley *et al.* 1968). Increasing the application rate increased the amount of trifluralin which volatilized, while increasing the volume of the diluent from 2.34 kl/ha to 300 kl/ha decreased vaporization by 50 percent. Another practice which decreased vapor losses was percolating trifluralin into dry soil in enough water to moisten the soil, rather than spraying onto moist soil and then soil-incorporating it. Regardless of the mode of application, vapor losses in the first twelve hours were less than five percent of the trifluralin applied (Swanson and Behrens 1972). Spencer and Cliath (1974) stressed that volatilization, rather than degradation, accounted for losses of trifluralin activity after surface application, but Soderquist and co-workers (1975) considered photolysis to precede volatilization.

When trifluralin was applied to sand in the aquatic-terrestrial model ecosystem, the trifluralin was sand-adsorbed and did not enter water in algicidal quantities (Sanborn 1974).

Into organisms--Neither trifluralin nor nitralin are taken up by foliage (WSSA 1974). When trifluralin was tested in the aquatic terrestrial ecosystem, sand-applied trifluralin resulted in greater accumulation of the herbicides by snails and fish than did foliage-applied trifluralin (Sanborn 1974). Caterpillars (*Estigmene acrea*) feeding on contaminated sorghum both distributed and degraded the trifluralin more effectively than snails (*Physa* sp.); the latter were unable to degrade trifluralin (Sanborn 1974). Metcalf and Sanborn (1975) judged trifluralin to accumulate in aquatic food chains to the same extent as methoxychlor. The ecological magnification level of 926 in fish (*Gambusia affinis*) and of 17,872 in snails was well correlated with its low water solubility and high lipid solubility. Nevertheless, 11 degradation products of trifluralin were found in the water of the ecosystem (Metcalf and Sanborn 1975). Model ecosystem data for nitralin were not available.

Persistence:

Trifluralin was found in 12 percent of soils with a history of regular trifluralin use (Stevens et al. 1970) and in 3.5 percent of 1729 samples tested for trifluralin under the national soils monitoring program in 1969 (Wiersma et al. 1972). The major losses of trifluralin and nitralin appear to be due to their volatilization, since an air flow of 0.04 m^3/hour over loamy sand soil was found to dissipate all the nitralin and over 75 percent of the trifluralin (Parochetti and Hein 1973). Horowitz (1969) characterized trifluralin as moderately persistent, with a greater persistence under field conditions than in greenhouses. The relative persistence of several herbicides under field conditions was: simazine=diuron > trifluralin=atrazine > fluometron > prometryne. Under greenhouse conditions the order was: simazine=diuron > atrazine > fluometron=trifluralin=bromacil=noruron > prometryne=ametryne > pyrazon (Horowitz 1969). The persistence of nitralin and trifluralin in soils is shown in Tables 24 and 25 for those data which could be quantified.

Table 24. PERSISTENCE OF NITRALIN IN SOIL

% Residue	Test # days	% loss/day	Conditions	Reference
0	900	0.11	bioassay	Miller *et al.* 1975
~0	450	0.22	bioassay	Ibid.
0	160	0.63	150 field survey	Savage 1973
0	~200	0.50	irrigated soils, bioassay	Isom *et al.* 1970

Table 25. PERSISTENCE OF TRIFLURALIN IN SOIL

% residue	Mean # days	% loss/day	Conditions	Reference
>0	900	<0.11	California	Lange *et al.* 1968
0	900	0.11	bioassay	Miller *et al.* 1975
5	730	0.13	bioassay, clay loam	Burnside 1974
1	730	0.14	Idaho, 1 lb/A	Parka and Tepe 1969
0.2	730	0.14	Tennessee, 5 lb/A	Ibid.
11	547	0.16	soil and org. matter	Weber *et al.* 1974a, 1974b
2	547	0.18	soil and clay	Ibid.
0	413	0.24	1.5 x ag levels	Bryant and Andrews 1967
30.7	365	0.19	Illinois, 0.75 lb/A	Parka and Tepe 1969
24.0	365	0.21	S. Carolina, 0.5 lb/A	Ibid.
13.3	365	0.24	California, 1 lb/A	Ibid.
10.0	365	0.25	Nebraska, 1 lb/A	Ibid.
0	260	0.38	irrigated soils, bioassay	Isom *et al.* 1970
0.21	252	0.40	3-4" incorporation, 4 lb/A	Savage *et al.* 1969
0.19	252	0.40	1-2" " 4 lb/A	Ibid.
0.02	252	0.40	surface applied, 4 lb/A	Ibid.
60	180	0.22	dried and mixed soil	Horowitz *et al.* 1974
50	180	0.28	undisturbed soil	Ibid.
0	160	0.63	250 field survey	Savage 1973
0	120	0.83	bioassay	Davis *et al.* 1964

When trifluralin was applied to sandy loam, persistence increased with increasing depth of soil incorporation (Menges and Tamez 1974, Burnside 1974) and with increasing levels of application (Savage and Barrentine 1969). In unsterilized Italian soil, nine to ten percent organic matter was most conducive to trifluralin persistence, but of soils sterilized by propylene oxide, clay or calcareous soils retained trifluralin longest (Petrosini *et al.* 1970). Savage (1973) surveyed 250 fields and concluded that trifluralin persisted equally well after repeated applications as after the first, whereas nitralin persistence decreased with repeated applications. Nitralin persisted longer in soil which had a lower pH.

Effects on Non-Target Species

Microorganisms--The data in Tables 26 and 27 show that, except at massive levels, trifluralin does not inhibit soil processes, soil bacteria, or soil fungi drastically. No data for its effects on algae were available. Noguchi and Nakazawa (1971) ranked eight herbicides with respect to their effects on soil nitrification. The order was: prometryne < lenacil < diphenamid < trifluralin < vernolate < MCPA < SWEP < PCP. Nitralin at 25 ppm stimulated the growth of *Pseudomonas fluorescens* while the same level of trifluralin had no significant stimulatory or inhibitory effect (Breazeale and Camper 1972). In meadow marshy soil and humus-peat-gley soil, however, nine kg/ha (4.1 ppm) and 30 kg/ha (14 ppm) were somewhat toxic (Tyunyaeva *et al.* 1974). Khikmatullaeva and Ibragimova (1973) reported that water levels of two ppm inhibited oxygen consumption, nitrification, and ammonification in reservoirs.

Invertebrates--The 24-hour LC_{50} for aquatic arthropods ranged from 8.8 ppm in the amphipod *Gammarus lacustris* to 13 ppm for stoneflies (*Pteronarcys* sp.). The 48-hour LC_{50} for the stonefly was 4.2 ppm, however, indicating cumulative effects. The LC_{50} for waterfleas (*Daphnia pulex*) was 0.24 ppm in 48 hours (Pimentel 1971). Data for nitralin were not available.

Table 26. EFFECTS OF TRIFLURALIN ON SOIL PROCESSES, ACTINOMYCETES, AND FUNGI

Parameter	Effect	Level Original	 kg/ha	Conditions	Reference
Respiration	–	5 T/A	1127	technical trifluralin	Stojanovic et al. 1972a
	+	5 T/A	1127	formulated Treflan	Ibid.
	+	≤960 ppm	≤2133	short-term	Whitworth et al. 1965
	–	≤960 ppm	≤2133	long-term	Ibid.
	–	15 x ag level		soil-incorporated	Ibid.
Ammonification	+	≤41 kg/ha	≤41	alluvial meadow soils	Bankov and Elenkov 1970
Nitrification	+	≤41 kg/ha	≤41	alluvial meadow soils	Ibid.
	0	6,12 ppm	13-26	silt loam	Thornsburg and Tweedy 1973
Actinomycetes	+	≤40 µg/g	88.9	soil	Curl et al. 1970
	0	3 kg/ha	3	soil	Tyunyaeva et al. 1974
	–	>9 kg/ha	>9	soil	Ibid.
Fungi	0	3 kg/ha	3	soil	Ibid.
	–	>9 kg/ha	>9	soil	Ibid.
	+	≤40 µg/g		soil	Curl et al. 1970
Fusarium oxysporum	+	≤40 µg/g	88.9	soil	Ibid.
Streptomyces	–	5 T/A	1127	soil	Stojanovic et al. 1972a
	–	5 T/A	1127	soil	Ibid.

+ = stimulation 0 = no effect – = inhibition

Table 27. EFFECTS OF TRIFLURALIN ON SOIL MICROORGANISMS AND BACTERIA

Parameter	Effect	Level: Original	Level: kg/ha	Conditions	Reference
Soil microorganisms	+	≤0.1%	≤2222		Rankov 1971
	–	>0.1%	≤4444		Ibid.
	0	≤15 kg/ha	15	leached chernozem	Gruzdev *et al.* 1973
Bacteria	–	5 T/A	11207	technical trifluralin	Stojanovic *et al.* 1972a
	–	5 T/A	11207	formulated Treflan	Ibid.
	+	<40 µg/g	<89.9		Curl *et al.* 1970
	0	3 kg/ha	3	meadow-marshy soil	Tyunyaeva *et al.* 1974
Cellulolytic	+	<41 kg/ha	≤41	alluvial meadow soils	Rankov and Elenkov 1970
	+			for 3-7 weeks	Rankov 1971
	–			heavy or alkaline soils	Ibid.
Nitrifying	–				Hamdi and Tewfik 1969b
Ammonia-fixing	0				Ibid.
Azotobacter	0				Ibid.
Bacillus	0	<50 µg/L	0.11	culture	Breazeale and Camper 1972
Pseudomonas	0	<50 µg/L	0.11	culture	Ibid.
Erwinia	0	<50 µg/L	0.11	culture	Ibid.

+ = stimulation   0 = no effect   – = inhibition

Fish and amphibians--The 24-hour LC_{50} of trifluralin for rainbow trout (*Salmo gairdneri*) was 0.21 ppm. For bluegills (*Lepomis macrochirus*) the LC_{50} decreased as time and temperature increased: the 24-hour LC_{50} at 45°C was 1.3 ppm while the 96-hour LC_{50} at 65°C was 0.13 ppm (Pimentel 1971). The 24-hour LC_{50} for Fowler's toad (*Bufo woodhousii fowleri*) tadpoles was 0.18 ppm (Pimentel 1971). Data for nitralin were not available.

Birds--The acute, oral LD_{50} of trifluralin for mallards and pheasants was greater than 2000 mg/kg (Tucker and Crabtree 1970); so was the acute oral LD_{50} for chickens (Pimentel 1971). Data for nitralin were not available, and chronic feeding data were available neither for nitralin nor for trifluralin.

Mammals--The acute oral LD_{50} of trifluralin to rats was greater than 10,000 mg/kg; for dogs and rabbits, greater than 2000 mg/kg; and for mice, greater than 5000 mg/kg (WSSA 1974). No data on chronic toxicity were available for trifluralin or nitralin. The acute oral LD_{50} of nitralin to rats was greater than 2000 mg/kg (Jones et al. 1968).

Conclusions:

Although trifluralin is not particularly toxic to mammals in single doses, it is highly toxic to fish, amphibians, and aquatic arthropods. It is, moreover, highly persistent and is magnified in aquatic food chains. The data are insufficient to predict disastrous consequences if large amounts of trifluralin are disposed of in soil, but also insufficient to be reassuring. Data for nitralin are too sparse to permit any conclusions.

Picloram

Picloram, the common name for 4-amino-3,5,6-trichloropicolinic acid, was introduced as Tordon by the Dow Chemical Co. in 1963 as a general herbicide for woody plants and broadleaf weeds. Pure picloram is a white powder with an odor similar to that of chlorine which decomposes at 215° C without melting. Its water solubility is 430 ppm and its vapor pressure is 6.16 x 10^{-7} mm mercury at 35°C. Formulations include the potassium salt in pellets or aqueous solution, the amine salts, and esters. The potassium salt has a water solubility of 400,000 ppm.

Degradation

Biological--The biological degradation of picloram has been assumed, since degradation ends when soil is sterilized (Parker and Hodgson 1966) and because a lag period precedes picloram degradation in soil (Grover 1967). The few reports of specific organisms which degrade picloram, or of degradative products, are shown in Table 28.

In heavy clay soil, degradation of picloram was preceded by a lag phase of seven days for 0.25 ppm, 30 days for 0.5 ppm, and 90 days for one ppm, with a soil half-life of 55, 90 and 180 days at the three levels. Differences in the soil half-life were attributed entirely to the variation in lag periods, with a one percent per day rate of degradation thereafter (Grover 1967). Degradation of one pound of picloram in soil required concomitant degradation of 10,000 to 100,000 pounds of organic matter (Youngson et al. 1967). The only products of picloram degradation which have been reported so far are the mono-dechlorinated and mono-demethylated compounds (Table 28), carbon dioxide, and chloride ion (Goring and Hamaker 1971, Meikle et al. 1974). Naik et al. (1972) considered the possibility of obtaining a readily biodegradable picloram to be remote, since most substitutions (decarboxylation, dechlorination at C_5, deamination) decreased phytotoxicity without affecting persistence. Adding

Table 28. DEGRADATION OF PICLORAM BY MICROORGANISMS

Organism	Product	Conditions	Reference
Unidentified	CO_2; Cl^-; 4-amino-6-hydroxy-3,5-dichloropicolinic acid		Meikle *et al.* 1973
	4-amino-3,5-dichloro-6-hydroxypicolinic acid; 4-amino-3,5,6-trichloropyridine	soil, wheat microorganisms	Goring and Hamaker 1971
	unidentified		Arnold *et al.* 1966
Unidentified	unidentified		Grover 1967

carboxyl, hydroxyl or amino groups increased persistence. Additional chlorine decreased persistence as did *alpha*- or *beta*-methylation, while *gamma*-methylation increased persistence.

The rate of picloram detoxification in soil may follow half-order kinetics (Hamaker *et al.* 1968), or first-order kinetics at low concentrations and Michaelis-Menten kinetics at high concentrations (Youngson *et al.* 1967, Grover 1967). Meikle *et al.* (1973) considered detoxification to follow 0.8-order kinetics rather than first-order kinetics, and Hance and McKone (1971) concluded that neither zero-order, half-order, first-order or Michaelis-Mentin kinetics applied.

The sum of these data on the degradation of picloram in soil is that, although the herbicide is undoubtedly detoxified in soil, active microbial degradation has not been demonstrated. Cometabolism of picloram by soil microorganisms is a plausible but unproven hypothesis, and degradation in soil probably proceeds no further than detoxification.

In cattle, feeding five ppm of picloram resulted in almost quantitative urinary elimination (Fisher *et al.* 1965). Milk residues did not exceed 0.2 ppm even when cows were fed 10,000 ppm picloram, but the milk was not tested for metabolites (Paulson 1975).

Photolytic--In water at alkaline pH, 12×10^{-2} M picloram (2898 ppm) exposed to 200 $\mu W/cm^2$ of ultraviolet light (253.7 nm) was 20 percent decomposed after 48 hours. Decomposition included destruction of the pyridine nucleus and release of two chloride ions per molecule. Sunlight was both less effective and less consistent in its effects (Hall *et al.* 1968). Hedlund and Youngson (1972) considered photolysis to follow pseudo-first-order kinetics for concentrations of less than 4.14×10^{-4} M (99.98 ppm) and to proceed at depths of 3.65 meters even in hazy sunlight. Mosier and Guenzi (1973) exposed 2.06×10^{-3} M of the sodium salt of picloram to light between 300 nm and 380 nm. The pH decreased from 9.5 to 6.0 in 36 hours and two chloride ions were released per molecule. The extremely limited CO_2 formation argued against decarboxylation as a major photolytic pathway. Baur, Bovey and McCall (1973) analyzed the effects of light and temperature on the degradation of picloram as the sodium salt and as the free acid. The acid decomposed significantly at 60°C and was not decomposed by UV light, whereas the salt was decomposed at 30°C, 60°C and by UV light at 356 nm (Baur *et al.* 1973).

Chemical and physical--Picloram was completely degraded by liquid ammonia in the presence of either metallic sodium or lithium (Kennedy *et al.* 1972a) but was unaffected by 9*N* or 18*N* sodium hydroxide (Kennedy *et al.* 1972b). In soils, Corbin and Upchurch (1967) observed no detoxification of picloram on an organic soil, but Hance (1967, 1969) considered chemical degradation of picloram in soils to be theoretically feasible.

When heated to 225°C in a muffle furnace, picloram lost 48 percent of its weight, but remained a white solid (Stojanovic *et al.* 1972b). When heated to 900°C, the volatile products of picloram degradation were carbon monoxide, carbon dioxide, chlorine and ammonia (Kennedy *et al.* 1972a, 1972b).

Transport:

Within soil--Adsorption of picloram decreased with increasing pH (Farmer and Aochi 1974, Helling 1971b) and followed a Freundlich isotherm, characteristic of physical adsorption between the herbicide and humic acid molecules (Khan 1973). Adsorption was greater with smaller soil aggregates at specific bulk densities, but pore-water velocity was more important than either aggregate size or pore-water velocity (Davidson and Chang 1972). Increasing organic matter increased picloram adsorption, whereas the amount of clay was of minor importance (Hamaker *et al.* 1966, Farmer and Aochi 1974). Adsorption decreased slightly with increasing temperature (Farmer and Aochi 1974). The relative adsorption of picloram on several adsorbents was: activated charcoal > anion exchange resins > peat moss > cellulose triacetate. No adsorption occurred on montmorillonite, kaolinite, cation exchange resins, wheat straw or cellulose triacetate (Grover 1971). Van Genuchten and co-workers (1974) stressed that picloram adsorption was not a single-valued function.

The amount of leaching observed for picloram after various times in different soils is shown in Table 29. Most picloram in a North Carolina soil remained in the top layer (7.5 cm) of soil. Less than 1.2 meter downslope movement occurred, probably because there were only low levels of runoff (Lutz *et al.* 1973). In Saskatchewan, most of the picloram remained in the top six inches (15 cm) of soil, although leaching increased as the soil organic matter decreased (Keys and Friesen 1968). When picloram levels in pasture soils were monitored, residues in the 24 to 36 inch layer (60-90 cm) of soil were greater one to three years after treatment than in the year of treatment, and dissipation from this layer was slow (Scifres *et al.* 1969). Deeper leaching occurred on a three percent slope than on a one percent slope (Scifres *et al.* 1971). Phillips and Feltner (1972) considered picloram presence at 2.4 meters to be the result of movement through soil cracks rather than by leaching. Among factors determining the extent to which picloram leached were the soil type, with greater leaching in sand than in clay loam (Baur *et al.* 1972) and the average pore-water velocity (Davidson and McDougal 1973). Both soil texture and the uniformity of soil pores affected leaching. Diffusion from conducting pores into adjacent micropores was suggested by Ping and co-workers (1975). Conventional sprays and polymerized sprays leached to the same extent (Baur *et al.* 1972) and adsorption was negatively correlated with diffusion (Walker 1970). Byrd and co-workers (1971) concluded that the lateral migration of 2.16 lbs/A (2.42 kg/ha) picloram sprayed on roadsides and railbeds would not result in "undue hazard" to adjacent vegetation.

Between soil and water--When 1.12 kg/ha of a one to one mixture of 2,4,5-T and picloram were sprayed on grassland soil in the Blacklands of Texas, groundwater was contaminated by one to four ppb of picloram two to nine months later, but not by 2,4,5-T (Bovey *et al.* 1975). When 2.24 kg/ha

Table 29. MOVEMENTS OF PICLORAM IN SOILS

Depth of leaching (cm)	time (days)	cm/day leached	Conditions	Reference
2400	<2190	>1.10	silty clay loam, 1.12 kg/ha	Phillips and Feltner 1972
≥96	1095	>0.08	pasture soils	Scifres *et al.* 1969
75	550	0.14	silty clay, 0.25 lb/A	Scifres *et al.* 1971
<244	365	<0.67	sand or clay loam	Baur *et al.* 1972a
≤90	<280	<0.32	light silt loam, 2 cm/h	Herr *et al.* 1966
≤75	<280	<0.27	light silt loam, 4 cm/h	Ibid.
<60	290	<0.21	sandy clay loam, 0.25 lb/A	Scifres *et al.* 1971
183	182	1.00	sand or clay loam	Baur *et al.* 1972a
≤90	101	<0.89	light silt loam, ≥8 cm/h	Herr *et al.* 1966
≤120	90	≤1.33	Puerto Rican forest soils	Dowler *et al.* 1968
≤60	56	≤1.07	soil	Brady 1975
60	42	1.43	sandy soil	Merkle *et al.* 1966
15	30	0.50	sand or clay loam	Baur *et al.* 1972a

of this mixture were applied five times at six month intervals, washoff from foliage accounted for most of the surface-water contamination. Residues on the grass were 50 to 70 ppm immediately after spraying, and if heavy rains occurred soon after, washoff contained 400 to 800 ppb picloram (Bovey *et al*. 1974). Davis and Ingebo (1973) estimated that 4.5 percent of the picloram applied to a chaparral watershed was lost to stream water. After a heavy rain, water contained 30 to 370 ppb, and water contamination occurred until 40 inches of rain had fallen over a 16 month period.

Surveys of grasslands water sources determined that 1.1 kg/ha sprayed over a 32 hectare area did not contaminate soil 0.8 km downslope from the treated area, nor was picloram detected in wells over a two year period. The dissipation of picloram from contaminated ponds proceeded at 14 to 18 percent per day at first, but ponds still contained 0.005 ppm after 100 days (Haas *et al*. 1971). Streams 1.6 km from picloram-treated plots contained detectable residues for up to eight months, with maximum water levels of 0.04 ppm (Baur *et al*. 1974). In the Ogalalla aquifer, picloram did not move 150 feet from the point of injection during a ten day period (Schneider *et al*. 1971).

Persistence:

Quantifiable data on persistence of picloram in soils are summarized in Table 30 and Table 31, the latter summarizing a single study of picloram persistence in soils from several states (Goring *et al*. 1965). Data for the disappearance of picloram in a sandy loam soil in Nova Scotia are shown in Figure 5.

The relative persistence of several herbicides one and two years after application to Puerto Rican forest soil was: fenac > prometone > picloram > diuron > bromacil > dicamba when phytotoxicity was the criterion.

After one year, residues from nine lbs/A (10.28 kg/ha) of picloram were less than ten percent as high as residues from 27 lbs/A (30.94 kg/ha)

Table 30. PERSISTENCE OF PICLORAM IN SOIL

% residue	# months	% loss per month	Conditions	Reference
>0	36	<2.78	silty clay loam	Phillips and Feltner 1972
0	42	2.38	semiarid rangeland	Scifres *et al.* 1971
25	12	6.25	0-9 inch layer, Saskatchewan	Keys and Friesen 1968
10	24	3.75	>8 cm., Saskatchewan	Ibid.
≤6	48	≥1.96	≤48 cm., Saskatchewan	Ibid.
10	16	5.62	3 oz/A, Saskatchewan	Ibid.
90	3.3	3.00	1 lb/A, N. Carolina	Lutz *et al.* 1973
50	>3.3	15.15	4.79 ppm, K-salt	Altom and Stritzke 1973
≤6	12	≥7.83	sprayed on grass	Getzendaner *et al.* 1969
≤52.5	36	1.32		Bereznovskii 1974
0.10	28.1	3.56	soil incorporated	Ragab 1975
>0	30	<3.33	California ag soils	Lange *et al.* 1968
>0	>4	<25.00	bioassay	Davis *et al.* 1964
0.05	4	24.99	bioassay, Bracken soils	Parker and Hodgson 1966
0.03	12	8.33	" " "	Ibid.
0.003	24	4.17	" " "	Ibid.

Table 31. DISAPPEARANCE OF TORDON HERBICIDE (PICLORAM) FROM SOILS: DATA FROM GORING, YOUNGSON, AND HAMAKER 1965

% residue	Time (months)	Loss/month (%)	Half-life* (months)	Location
0	42	2.44	<5	California
0	41	2.44	<5	"
22	28	2.78	13	"
17	28	2.96	11	"
0	28	3.57	<3	"
0	25	4.00	<3	Kansas
8	23	4.00	3	California
0	23	4.35	<2	"
13	22	3.95	8	Kansas
9	17	5.35	5	South Dakota
9	16	5.69	5	Minnesota
29	13	5.46	7	"
42	8	7.25	6	California
4	5	19.20	1	Kansas
9	5	18.20	1	South Dakota
20	4	20.00	2	Minnesota

*Authors' estimate

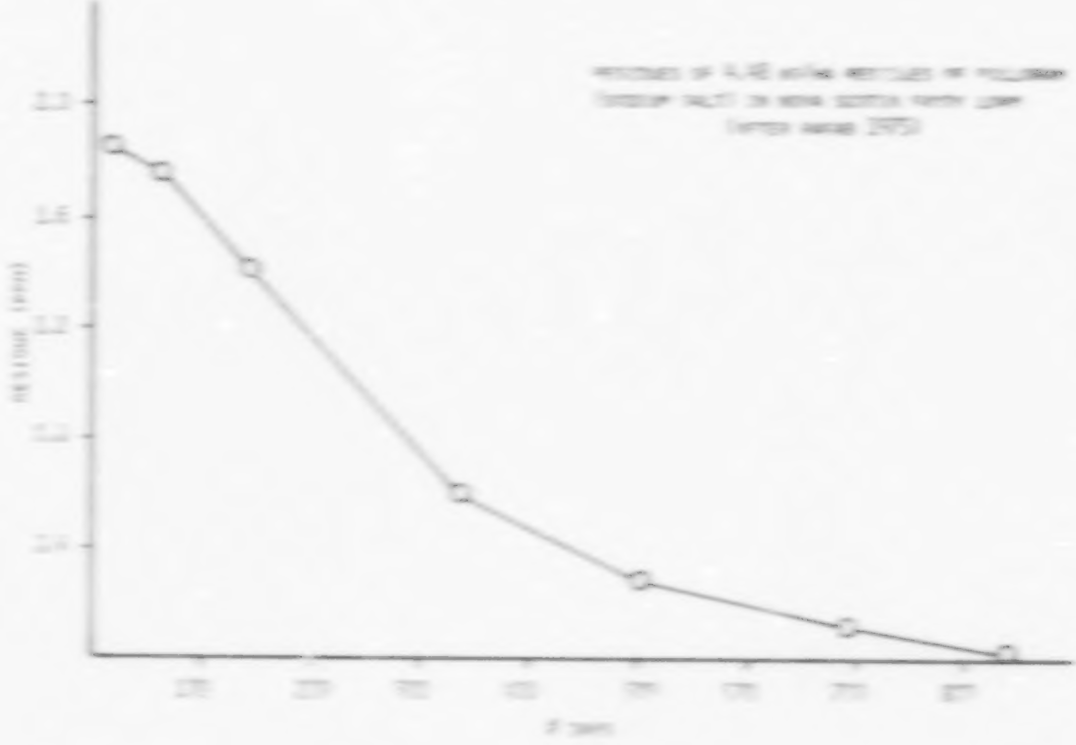


Figure 5

(Dowler *et al.* 1968). After three years, Parker and Hodgson (1966) observed no further decrease in residues from four lbs/A (4.48 kg/ha) picloram in soil underlying bracken and residues were still toxic to beans grown in pots.

Picloram persistence increased with increasing soil depths (Herr *et al.* 1966, Scifres *et al.* 1969, Hunter and Stobbe 1972) and with cool weather at the time of application (Scifres *et al.* 1969). Residues from Tordon beads exceeded those from Tordon liquid (Getzendaner *et al.* 1969) and residues from conventional sprays and from granules exceeded those from polymerized sprays (Baur *et al.* 1972). Picloram disappeared more quickly from cleared forest soil than from litter-covered forest soil (Brady 1975). The persistence of picloram in water exposed to hazy sunlight was greater than 30 days in shallow containers, and greater than 56 days in deep containers (Hedlund and Youngson 1972).

Effects on Non-Target Species:

Microorganisms--The effects of massive levels of picloram on numbers of bacteria in soil are negative: five tons per acre (5000 ppm) of picloram incorporated into a loam soil inhibited bacterial growth for at least 56 days whether the herbicide was pure or formulated (Stojanovic *et al.* 1972a). Debona and Audus (1970) reported bacterial inhibition at less than 150 ppm in soil columns, but 1000 ppm was reported to have no effect on soil bacteria (Beynon *et al.* 1966) and 100-fold the normal agricultural levels neither stimulated nor depressed bacterial growth for six months (Van Schreven *et al.* 1970). In Oregon soils, Tu and Bollen (1969) demonstrated that picloram inhibited bacterial numbers at ten ppm, but not at one ppm.

Similarly discordant data have been reported for individual species of bacteria: *Azotobacter* was inhibited by 100-fold the usual agricultural levels (Van Schreven *et al.* 1970) but not by 1000 ppm (Beynon *et al.* 1966), and *Bacillus subtilis* was inhibited by one ppm (Thomas *et al.*

1973). Inhibition of *Pseudomonas fluorescens* in culture occurred at 50 ppm picloram (Breazeale and Camper 1972). Unaffected by 1000 ppm picloram when cultured on agar were: *Nocardia opaca*, *Pseudomonas aeruginosa*, *Rhizobium phaseoli*, *Aerobacter* and *Aerogenes* (Beynon et al. 1966).

Fungal growth in soil was stimulated by 5000 ppm analytical grade picloram for 56 days (Stojanovic et al. 1972a) and unaffected on agar by 1000 ppm (Beynon et al. 1966). Streptomycetes, however, were inhibited by 5000 ppm under the same conditions which stimulated fungal growth (Stojanovic et al. 1972a). On agar, no significant effects of up to 1000 ppm were seen in *Aspergillus terreus*, *Fusarium oxysporum*, *Penicillium digitatum*, *Rhizoctonia solani*, *Pythium ultimum*, *Trichoderma lignorum*, *Verticillium alboatrum*, *Streptomyces scabies*, or *Aspergillus niger* (Beynon et al. 1966). In soil, formulated picloram at one and ten ppm inhibited the growth of *Mucor* and *Penicillium* species, and stimulated *Aspergillus* and *Terreus* species (Beynon et al. 1966).

Such differences in toxicity due to differences in species and/or experimental techniques were not found for the effects of picloram on algae. One ppm did not inhibit the growth of *Chlorella pyrenoidosa* on agar (Beynon et al. 1966) but 50 ppm depressed growth of *Chlorella vulgaris*, *Chlorococcum*, and *Cylindrospermum licheniformis* in culture for up to 542 days. Picloram inhibited soil algae more strongly than did 2,4-D (Arvik et al. 1971). A number of fungi (*Trichoderma viride*, *Fusarium oxysporum*, *Helminthosporium victoriae*, *Penicillium lanosum*, *Aspergillus flavus*) grew on up to 1000 ppm picloram but could not use it as their sole source of either carbon or nitrogen (Hameed and Foy 1974). The relative effects of several herbicides on soil nitrification were: dichlobenil < paraquat < picloram < *2,3,6*-TBA = chlorthiamid < bromoxynil < chlorfluorazole < ioxynil < propanil (Debona and Audus 1970). The effects of picloram on soil processes are shown in Table 32, and are seen to be minor at low levels but inhibiting at high levels.

Table 31. EFFECTS OF PICLORAM ON SOIL PROCESSES

Parameter	Effect	Level Original	 PPM	# days observed	Conditions	Reference
Respiration	0	1 ppm	1	172	soil	Groover 1972
	+	100,300 ppm	100,300	172	soil	Ibid.
	0	1000 ppm	1000		soil	Beynon et al. 1966
	+	240 ppm	240		soil	Whitworth et al. 1965
	-	480 ppm	480		soil	Ibid.
	+	5 T/A	5000	56	analytic. std.	Stojanovic et al. 1972a
	+	5 T/A	5000	56	formulated	Ibid.
	0	10 x ag levels		56	sandy loam	Van Schreven et al. 1970
	-	10 x ag levels		<24	sandy loam	Ibid.
Ammonification	0	≤100 x ag levels		182	sandy loam	Ibid.
	0	1000 ppm	1000			Beynon et al. 1966
Nitrification	0	≤100 x ag levels			sandy loam	Van Schreven et al. 1970
	-					Tu and Bollen 1969
	0	1000 ppm	1000		soil	Beynon et al. 1966
	0	0.07 kg/ha	0.03	20-40		Bezuglov et al. 1973
	-	2 ppm	2		weak inhibition	Dubey 1969
	-	20-100 ppm	20-100	56	Complete inhibition in poor soils	Ibid.
	0	6-12 ppm	6-12		silt loam	Thornburg and Tweedy 1973
	-	150	150		50% inhibition	Debona and Audus 1970
	-	500	500		100% inhibition	Ibid.

+ = stimulation   0 = no effect   - = inhibition

Analytical grade picloram, incorporated in soil at 5000 ppm (five tons per acre) stimulated fungal growth but inhibited both streptomycetes and bacteria. Formulated picloram inhibited bacteria and streptomycetes, but was not tested against fungi (Stojanovic *et al.* 1972a).

Invertebrates--Levels of one ppm did not affect *Daphnia* over a ten week period (Lynn 1965). The 48-hour LC_{50} of picloram for an amphipod (*Gammarus lacustris*) was 48 ppm, and the 24-hour LC_{50} for the same species was 50 ppm. For stone fly nymphs, the 24-hour LC_{50} was estimated to be 120 ppm (Pimentel 1971). Snails survived 380 ppm but not 530 ppm (Lynn 1965) and the growth of eastern oysters (*Crassostrea virginica*) was not affected by one ppm (Butler 1965).

Fish and amphibians--The 24-hour LC_{50} for various fish species, reviewed by Kenaga (1969) ranged from 2.2 ppm of the isooctyl ester for channel catfish (*Ictalurus punctatus*) to less than 36 ppm of the acid for goldfish (*Carassius auratus*). Tolerance generally increased with increasing temperature (Pimentel 1971). Sergeant and co-workers (1970) noted that commercial formulations of picloram were toxic to fish at concentrations at which picloram itself was not. They suggested that 2-(3,4,5,6-tetrachloro)-2-pyridylguanidine might constitute a highly toxic impurity.

Birds--The acute oral LD_{50} of picloram was over 2000 mg/kg in young mallards and young pheasants (Tucker and Crabtree 1970) and the five-day LC_{50} was greater than 5000 ppm for bobwhite, Japanese quail, Pheasants and mallards (Heath *et al.* 1970). Somers and co-workers (1973, 1974a, 1974b, 1974c) observed no detrimental effects on hatching success in pheasants or chickens when eggs were sprayed with picloram and/or 2,4-D and 2,4,5-T. This study is discussed under the section on 2,4-D and 2,4,5-T.

Mammals--The acute oral LD_{50} for rats was 8200 mg/kg (Pimentel 1971). Death occurred within 15 days for half the rats fed 8200 mg/kg, for half the mice fed 2000 to 4000 mg/kg, and for half the rabbits fed 2000 mg/kg (Lynn 1965). Picloram was tumorstatic in mice (Bradley *et al.*

1974). Levels of up to 1000 mg/kg did not affect the maternal weight gain, litter size or resorption rate in pregnant rats if dams who died were excluded from the data, but fetal ossification was inhibited (Thompson *et al*. 1972). McCollister and Young (1969) reviewed data on the toxicology of picloram and noted that 0.1 percent (100 ppm) did not affect murine reproduction and that 3000 ppm fed to three generations of rats was also without reproductive effect.

Conclusions:

The sparse data on the biological degradation of picloram and on the nature and persistence of its terminal residues argue against its disposal in soil.

In all probability the effects of picloram on soil processes and soil microorganisms would vary sharply with soil, temperature and other environmental factors as well as with species. The most probable consequence of picloram contamination would therefore be a more or less subtle shift in the balance of soil-dwelling species. Since picloram persists for long periods in soil, the alterations would be of long duration if not permanent.

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SECTION IV INSECTICIDES

CHLORINATED HYDROCARBON INSECTICIDES

Of the numerous pesticides commonly included under the heading of "chlorinated hydrocarbons" or "organochlorines", the 12 compounds to be discussed are: *DDT*, its hydroxy-analog *dicofol*, and its methoxy-analog *methoxychlor*; the cyclodiene insecticides *aldrin*, *dieldrin*, *endrin*, *mirex*, *kepone (chlordecone)*, *heptachlor*, and *chlordane*; the sulfur containing cyclodiene, *endosulfan*; and the chlorinated terpene mixture designated as *toxaphene*.

The literature for each compound or group of compounds is reviewed separately, but certain papers which compare the chlorinated hydrocarbon insecticides with each other and with other pesticides are reviewed below. In this way a perspective is obtained as to the relative stability, mobility, persistence, and toxicity of these highly controversial compounds. Similarly, conclusions for all the organochlorine insecticides are discussed at the end of the section and not after the separate reviews.

Degradation:

The ultimate fate of the millions of tons of chlorinated hydrocarbon insecticides which have been dispersed into the air, the soil, and the water is not known. Schuphan and Ballschmiter (1972) concluded that the dechlorination of the cyclodienes under the influence of the soil pH was essentially impossible, but considered photochemical dechlorination, as on leaf surfaces, of possible significance. Alexander (1968) argued persuasively that biodegradation of certain molecules, including DDT, might occur only in the laboratory, since cometabolism is of no advantage to microorganisms outside the laboratory (c.f. DDT, section on biological degradation).

While the ultimate reality of such hypotheses remains to be tested, the persistence of most chlorinated hydrocarbon insecticides is demonstrated by the sizable residues present 16 and 21 years after application (Nash and Harris 1973, Bennett *et al.* 1974, Kuhr *et al.* 1972), and by the presence of chlorinated hydrocarbon residues in all phases of the environment: in soils (Gish 1970, Wiersma *et al.* 1972), in rainwater (Wheatley and Hardman 1965, Tarrant and Tatton 1968) and groundwater (Achari *et al.* 1975), in the atmosphere (Risebrough *et al.* 1968, Stanley *et al.* 1971, Bidleman and Olney 1974) and in polar mammals (Tatton and Ruzicka 1967, Clausen *et al.* 1974).

Wiersma *et al.* (1972) examined fifty sites in each of five cities for arsenic, DDT and its metabolites, dieldrin, chlordane, heptachlor and its epoxide, toxaphene, endrin, and organophosphates. Considerable variation was found between cities, with Miami having the highest residues and Houston the lowest; within cities, residues were higher in lawns than in unkempt areas. Organophosphate residues were rare, but organochlorine residues were common.

The levels of pesticide residues found in three surveys of agricultural soils are shown in Table 33.

Transport:

Although the organochlorines are among the least mobile of insecticides, movement through soil is seen during their long persistence in soil. The relative mobilities of organochlorine pesticides are shown in Table 34.

Burkhardt and Fairchild (1967) observed that the mobility of aldrin, heptachlor, and three organophosphate insecticides varied with the compound, its rate of application, the soil type, soil moisture, time of

Table 33. RESIDUES OF PESTICIDES FOUND IN SOILS

Compound	PPM 1/	PPM 2/	%	% 3/
DDT	97.70	1-9	45	*
DDE	12.65	*		*
DDD	3.33	*		*
Aldrin	2.13	0.75	37	75
Dieldrin	3.33	"	"	"
Endrin	6.55	*		*
Heptachlor	0.40	0.06	9	*
Heptachlor epoxide	0.24	"	"	*
γ-chlordane	0.63	0.86	9	*
BHC	*	*		75
Malathion	*	*		43
Atrazine	*	*		36
2,4,5-T	*	*		24

*Not sought

1/ S.W. Ontario farmland by Harris and Sans 1971.

2/ Canadian Atlantic Provinces by Duffy and Wong 1967.

3/ Five W. Alabama Counties by Albright et al. 1974.

Residues are given in PPM for contaminated soil in studies 1 and 2, and the percent of soils contaminated is shown for studies 2 and 3.

Table 34. RELATIVE MOBILITIES OF PESTICIDES IN SOIL: PESTICIDES LISTED IN DECREASING ORDER OF MOBILITY

1	2
BHC	Aldrin
Isodrin	Dyfonate
Heptachlor	Lindane
Endrin	Dieldrin
Toxaphene	Parathion
Dieldrin	DDT
Aldrin	Diazinon
Dilan	Azinphosmethyl
Chlordane	

1. Nash and Woolson 1968: mobility within soil.
2. Lichtenstein and Schulz 1970: volatilization in soil-water mix.

year and interval of sampling. Using bioassay of field-treated soils, they concluded that aldrin and heptachlor were more mobile than diazinon, parathion, or phorate. Nash and Woolson (1968) found aldrin, dieldrin, dilan, toxaphene and chlordane to be less evenly dispersed through cultivated sandy loam than were BHC or isodrin. After fourteen years, the greatest concentrations of dieldrin, dilan, toxaphene, and chlordane were found in the soil at depths between 7.6 and 23 centimeters. Except for BHC and isodrin, 95 percent of the pesticides remained in the upper 23 centimeters. Harris and Sans (1969) noted that organochlorines penetrated cultivated soil more deeply than uncultivated soil, and mineral soil more deeply than muck. Carter and Stringer (1970) found that aldrin, dieldrin, *gamma*-chlordane and heptachlor penetrated soils to the same extent, with soil type and soil moisture the chief factors affecting depth of penetration. In sandy soil, moisture affected penetration more than in muck, which retained 70 percent of all the pesticides in the upper soil layers regardless of moisture levels. Lichtenstein et al. (1971b) observed that cultivation sharply decreased soil residues of both heptachlor and aldrin (or dieldrin), presumably because cultivation increased volatilization. For heptachlor, a residue reduction of 76 to 82 percent was recorded.

Goerlitz and Law (1974) noted that the distribution of chlorinated hydrocarbons in stream bottom material varied significantly with size, organic matter content, and composition of the stream-bottom particles: sand was free of organochlorine residues while gravel with shell material carried large amounts of hydrocarbons. Ju-Chang and Liu (1970) compared the behavior of three organochlorines on three clays, and found the relative levels of adsorption to be: DDT > dieldrin > heptachlor. All three pesticides were adsorbed almost instantaneously to the surface of kaolinite and illite; and DDT and heptachlor diffused, somewhat more slowly, into the intralamellar spaces of montmorillonite. Dieldrin did not diffuse into montmorillonite. The difference was considered due to hydrogen-bond formation in the adsorption of DDT and heptachlor onto clays, while dieldrin adsorption was attributed to interactions of its epoxide ring with oxygen molecules in the clays. The adsorptive capacities of the clays with respect to these three pesticides was not correlated with their ion exchange capacities or with their specific surface areas. Contamination of drinking water by DDT and DDE was decreased by routine water treatment, but BHC and toxaphene were less readily removed, apparently because the latter are carried in solution rather than adsorbed on particulate contaminants (Nicholson et al. 1968).

Mistric and Gaines (1953) found the relative loss of insecticide from cotton leaf surfaces to be greatest for dieldrin and least for gamma-BHC, aldrin, dieldrin, heptachlor, and methyl parathion; losses of toxaphene, endrin and EPN were intermediate. That a large portion of the volatilized pesticides is subsequently precipitated is suggested by the presence of pesticides in rainwater and on unsprayed terrain. Hartley (1968) suggested, however, that those pesticide residues which escape to a height of 50 meters might, by eddy diffusion, be carried to the ionosphere and there undergo photolytic degradation.

Persistence:

Harris (1969) classified nine soil-applied pesticides as highly, moderately, or slightly residual: The highly residual compounds, which retained full activity for 48 weeks in sandy loam, were DDT and dieldrin; aldrin, Dasanit, and carbofuran were moderately residual, retaining full activity for 16 weeks in sandy loam; parathion, diazinon, phorate and Dursban lost all detectible activity within four weeks in sandy loam and were considered to be only slightly residual. The criterion of "activity" was defined by bioassay, beginning with four times the LD_{50} of each compound. The position of aldrin as "moderately persistent" resulted from its conversion to the less potent dieldrin. Tests in muck resulted in slightly longer persistence for all compounds.

Table 35 shows the relative persistence of the most common chlorinated hydrocarbon pesticides with respect to each other and to other pesticides. With the exception of methoxychlor, which was less persistent than parathion (Carlo et al. 1952), chlorinated hydrocarbons are consistently more persistent than insecticides of other classes.

Toxicity--

The relative toxicities of organochlorine pesticides in mammals and fish are listed in Table 36.

Macek and McAllister (1970) stated that, as a group, organochlorine insecticides are more toxic to fish than either the organophosphate or carbamate pesticides. Moreover, whereas large interspecies variability was observed in piscine sensitivity to organophosphates, relatively uniform sensitivities to organochlorines were found.

Table 35. RELATIVE PERSISTENCE OF PESTICIDES IN SOIL AS DETERMINED IN SEVEN STUDIES INCLUDING CHLORINATED HYDROCARBON INSECTICIDES. PESTICIDES LISTED IN DECREASING ORDER OF PERSISTENCE

1	2	3	4*	5	6	7
Toxaphene	Dieldrin	DDT	Heptachlor	BHC	Lindane	Toxaphene
Endrin	Heptachlor epoxide	Dieldrin	Diazinon	Dieldrin	DDT	Parathion
Aldrin	Aldrin	Chlordane	DDT	DDT	Aldrin	Methoxychlor
Dieldrin	DDD	Aldrin	Parathion	Chlordane	Parathion	
Isodrin	DDT	Heptachlor		Parathion		
Dilan	Endrin					
Heptachlor	Heptachlor					
Chlordane	Lindane					
BHC						

*Persistence determined by bioassay.

1. Lash and Harris 1973
2. Hill and McCarty 1967
3. Wiese and Basson 1966
4. Harris 1967
5. Wylie 1956
6. Beran and Guth 1965
7. Carlo et al. 1952

Table 36. RELATIVE TOXICITIES OF PESTICIDES TO MAMMALS, FISH, AND BEES: PESTICIDES LISTED IN DECREASING ORDER OF TOXICITY

Rats[1]	Fish[2]	Fish[3]	Bees[4]
Endrin	Endrin	Endrin	Azinphos
Dieldrin	Rotenone	*p,p'*-DDT	DDVP
Aldrin	Toxaphene	Dieldrin	Carbaryl
Toxaphene	Pyrethrum	Aldrin	DDT
Heptachlor	Aldrin	Dioxathion	Dicofol
Endosulfan	DDT	Heptachlor	
DDT	Lindane (*gamma*-BHC)	Lindane	
Chlordane	Heptachlor	Methoxychlor	
Dicofol	Parathion	Phosdrin	
Methoxychlor	Systox	Malathion	
	Dicofol	DDVP	
	Vapam	Methyl parathion	
	Malathion		
	delta-BHC		
	Diazinon		
	Perthane		
	Ovotran		
	Metasystox		
	alpha-BHC		
	Tedion		

1. Martin 1968: acute oral LD_{50} in rats.
2. Adlung 1957: Toxicity of pesticides to the guppy, *Lebistes reticulatus*.
3. Eisler 1970b: 96-hour toxicity in estuarine fish.
4. Anderson and Atkins 1958.

DDT and Dicofol

DDT is the common name for the technical product in which *p,p'*-DDT is the predominant compound. The acronym DDT stands for dichlorodiphenyltrichloroethane, known as 1,1,1-trichloro-2,2-bis-(4-chlorophenyl)ethane. DDT was the first of the wide spectrum organic insecticides, introduced in 1942 by Geigy under the trade names Gesarol, Guesarol, and Neocid. It is a potent nonsystemic stomach and contact insecticide which had little effect on phytophagous mites, aphids, or scale insects, but was used with striking success on a wide variety of agricultural and public health pests.

DDT is synthesized by the condensation of one mole of chloral with two moles of chlorobenzene in the presence of sulfuric acid. The *p,p'*-isomer forms colorless crystals with a melting point of 108.5^{0}C and a vapor pressure of 1.9 x 10^{-7} mm mercury at 20^{0}C. The technical product contains up to 30 percent of the *o,p'*-isomer and is a waxy solid of indefinite melting point. Pure *p,p'*-DDT and its *o,p'*-isomer are practically insoluble in water (0.0012 ppm at room temperature), moderately soluble in hydroxylic and polar solvents, and readily soluble in most aromatic and chlorinated solvents (Martin 1968). Brooks (1974, vol. 1) listed the solubility of DDT in acetone, benzene, and carbon tetrachloride as 580, 780, and 450 mg/ml, respectively.

Dicofol is the common name for 2,2,2-trichloro-1,1-bis-(4-chlorophenyl) ethanol, a nonsystemic acaricide with little insecticidal activity, introduced by Rohm and Haas in 1955 as Kelthane. It is synthesized by the reaction of 1,1-dichlorophenyl-1,2,2,2-tetrachloroethane with silver acetate followed by hydrolysis of the resulting ester. Commercially, dicofol is formulated as an 18.5 percent or 42 percent emulsifiable concentrate, an 18.5 percent wettable powder, or in a dust base at 30 percent. The wettable powder formulations are sensitive to solvents and surfactants, which may affect acaricidal activity or phytotoxicity.

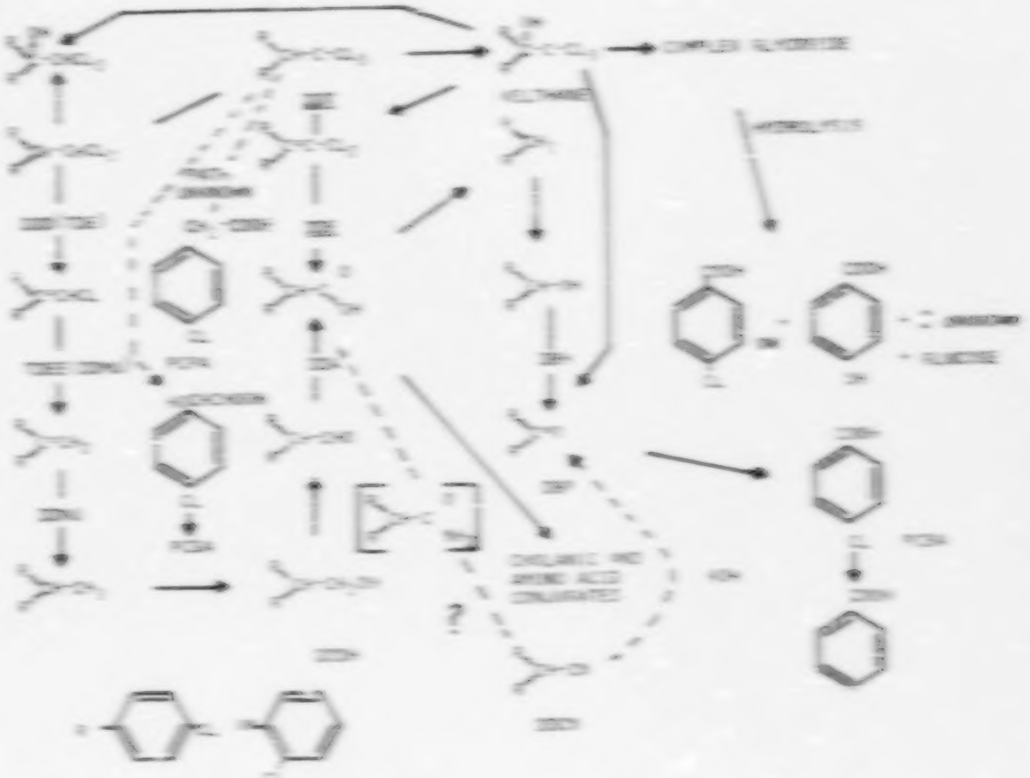
Pure dicofol is a white solid with a melting point of 78.5 to 79.5°C, but the technical product is a brown viscous oil of about 80 percent purity, essentially insoluble in water and soluble in most aliphatic and aromatic solvents. Dicofol is also a metabolite of DDT (Khan 1975).

DDD is the acronym for dichlorodiphenyldichloroethane, known as 1,1-dichloro-2,2-bis(*p*-chlorophenyl)-ethane. It is both a metabolite of DDT (Brooks 1974, vol. 2) and an insecticide produced by Rohm and Haas as Rhothane for use against lepidoptera and as a mosquito larvicide. DDD is also referred to as TDE.

Degradation—

Biological--The major degradative pathways for DDT are dehydrodechlorination to DDE and reductive dechlorination to DDD (TDE), as shown in Figure 6. Some microorganisms and some insects are able to oxidize DDT to dicofol, but for mammals, plants, birds, and most microorganisms, DDD and DDE are the first, and often the only, metabolites. DDE is usually considered less acutely toxic than DDT, but it is more toxic to pigeons (Brooks 1974, vol. 2); moreover, DDE is extremely stable in most organisms, with a half-life of 250 days in pigeons (Bailey et al. 1969a, 1969b). DDD is readily metabolized in vertebrates via DDMU, as shown in Figure 6.

The metabolism of DDT by microorganisms is summarized in Tables 37 and 38, which show the products of DDT metabolism as well as the organism or conditions under which conversion occurred. Anaerobically, DDT was converted principally to DDD (Guenzi and Beard 1967); addition of alfalfa components accelerated the reaction, but two percent oxygen in the medium sufficed to inhibit the dechlorination (Burge 1971). Guenzi and Beard (1968) found that the anaerobic conversion of DDT to DDD


COMPLEX GLYCOSIDE
KELTHANE
HYDROLYSIS
GLUCOSE
CHOLANIC AND
AMINO ACID
CONJUGATES
?

MAJOR METABOLIC PATHWAYS FOR DDT

Figure 6

Table 37. DEGRADATION OF DDT BY MICROORGANISMS UNDER LABORATORY CONDITIONS INCLUDING THE ORGANISMS AND THE PRODUCTS FORMED. STRUCTURES FOR NAMED COMPOUNDS ARE SHOWN IN FIGURE 6

Organism	Products	Reference
Actinomycetes	DDD	Chacko et al. 1966
Aerobacter aerogenes	DDD	Wedemeyer 1966, 1967
Baker's yeast	DDD	Ibid.
Proteus vulgaris	DDD	Ibid.
Fusarium oxysporum	DBH, DBP	Engst and Kujawa 1967
Escherichia coli	DDD	Langlois 1967
Trichoderma viride	DDD, DDE, Dicofol	Matsumura and Boush 1968
Aerobacter aerogenes	DDD	Plimmer et al. 1968
Mucor alternans	3 hexane soluble 2 water soluble	Anderson et al. 1970
Erwinia sp.	dechlorinated ethane moiety	Van Dijk et al. 1973
Streptomyces	dechlorinated ethane moiety	Ibid.
Ankistrodesmus amalloides	DDE, DDD	Neudorf and Khan 1975
Daphnia pulex	DDE	Ibid.

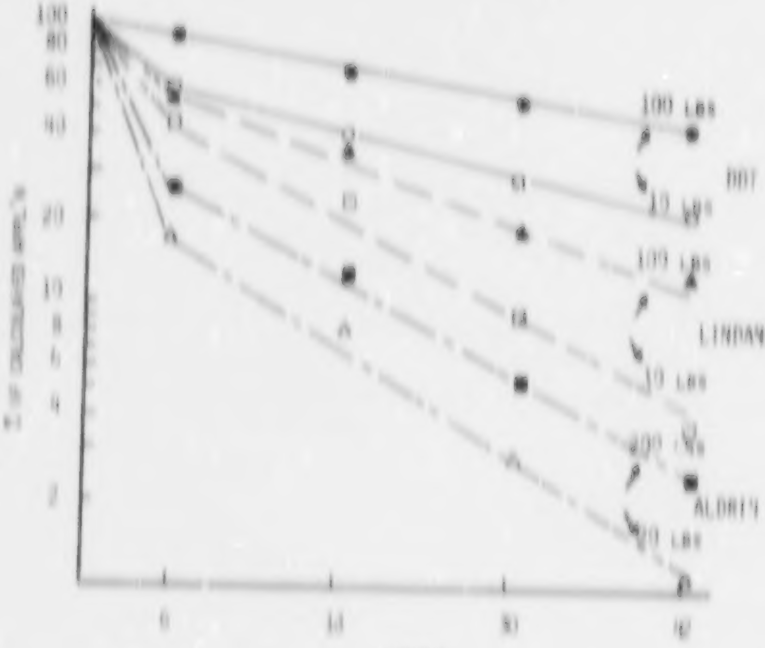
Table 38. DEGRADATION OF DDT BY MICROORGANISMS UNDER LABORATORY CONDITIONS INCLUDING THE CULTURE CONDITIONS AND THE PRODUCTS FORMED. STRUCTURES FOR NAMED COMPOUNDS ARE SHOWN IN FIGURE 6

Conditions	Products	Reference
Soil, anaerobic	DDD	Guenzi and Beard 1967
Soil, aerobic	DDE	Ibid.
20 cultures selected for dieldrin degradation	DDD, DDA	Patil and Matsumura 1968
Nitrogen, nitrogen + CO_2 atmosphere	DDD, DDE	Parr *et al.* 1970
Microarthropods	DDE	Butcher *et al.* 1970
Soil, anaerobic	DDD	Burge 1971
Lake Michigan microorganisms	TDE and DDNS	Matsumura *et al.* 1971
Estuarine films	TDE, DDNS, DDOH	Ibid.
Marine algae	TDE, DDNS, DDOH	Patil *et al.* 1972
Marine microorganisms	TDE, DDNS, DDOH	Matsumura and Boush 1972
Sewage	DDD, DDE, DBP	Pfaender and Alexander 1973
Flooded soil	DDD	Castro and Yoshida 1974
Flooded soil	DDD	Farmer *et al.* 1974
Everglades muck	DDE, DDD	Parr and Smith 1974

was essentially complete after 12 weeks, with only one percent of the DDT remaining, and only traces of metabolites other than DDD present. Even after six months, less than one percent of the ^{14}C-DDT had been converted to CO_2. Aerobically, degradation of DDT was much slower, and after six months 75 percent remained unchanged and four percent DDE and traces of DDD were found (Guenzi and Beard 1968). The loss of DDT, lindane, and aldrin from a Miami silt loam is shown in Figure 7.

Langlois (1967) found that *E. coli* were able to convert DDT to DDD, with the reaction 50 percent complete in two days and more than 90 percent complete in one week; skim milk inhibited the conversion. Fungi were found to be ineffective in converting DDT to DDD under laboratory conditions (Chacko et al. 1966). Under anaerobic conditions, DDT was converted to DDD more rapidly in an argon atmosphere than in a nitrogen atmosphere, which in turn was more effective than CO_2, which was more effective than CO_2 mixed with air (Parr et al. 1970). The authors suggested that CO_2 favored fungal growth and so retarded the growth of DDT degrading organisms. Both DDT and its analog, methoxychlor, were more effectively degraded in flooded anaerobic soil than in aerobic upland soils; DDD accumulated in the flooded soils (Castro and Yoshida 1971). Fresh water diatoms, collected from mosquito breeding sites in Winnipeg, were found to convert DDT to DDE (Miyazaki and Thorsteinson 1972).

Hydrogenomas species aerobically effected ring fission of the DDT analog *p,p'*-dichlorodiphenylmethane (DDM), but were unable either to cleave the DDT ring or to grow on DDM or on *p*-chlorophenylacetic acid (Focht and Alexander 1971). *Hydrogenomas* species were able, anaerobically, to carry out the conversion sequence: DDT → DDM → DBP, after which addition of oxygen and fresh cells resulted in ring cleavage and in the eventual formation of *p*-chlorophenylacetic acid, which can be degraded to CO_2. Alexander (1973) postulated that cometabolism, a process in which a compound is metabolized but not utilized by the degrading organism, is of no selective advantage to the organism, which



EFFECT OF CONCENTRATION ON LOSS OF ALDRIN, LINDANE, AND DDT FROM A MIAMI SILT LOAM; APPLICATION IN LB/A

LICHTENSTEIN AND SCHULZ, 1959b

Figure 7

derives no energy from the reaction. Therefore, even though it is possible to select organisms which cometabilize pesticides in the laboratory, it is not possible to select for such degradative reactions under field conditions, since cometabolizing organisms will not grow more rapidly in contaminated areas than organisms which do not cometabolize. Cometabolism is assumed if degradation occurs in a nonsterile medium but not in sterile medium and no organism is found which can use the substrate as its sole source of carbon (Pfaender and Alexander 1973). By this definition, sewage microorganisms cometabolized DDT to DDD, DDE, and DBP; adding glucose enhanced DDD production at the expense of DBP synthesis (Pfaender and Alexander 1973).

In an Everglades muck, DDT was slowly converted to DDD and DDE. Under the most favorable conditions, 10.1 percent of the DDT was converted to DDD, two percent to DDE, and 83.1 percent remained unaltered. Addition of lime to the muck favored the degradation of DDT, either by stimulating the growth of DDT-degrading organisms, or by favoring the desorption of DDT from the muck (Parr and Smith 1974). Both flooding and the application of organic matter shifted DDT degradation from formation of DDE to DDD (Farmer et al. 1974).

Mucor alternans in shake culture reportedly converted DDT into five metabolites, three of which were soluble in hexane and two in water. While the metabolites were not identified positively, they were not identical to DDD, DDE, DDA, DDP, dicofol, or 1,1-bis(*p*-chlorophenyl) ethane. The *M. alternans* spores, when placed in soil, were unable to degrade DDT (Anderson et al. 1970). The degradation of DDT in a cell-free *Aerobacter aerogenes* system suggested reduced cytochrome oxidase as the dechlorinating agent (Wedemeyer 1966).

In addition to the actual conversion of DDT by microorganisms, DDT was adsorbed by streptomyces, bacteria, and fungi (Chacko and Lockwood 1967). No particular difference in uptake was seen between fungi for any one pesticide, but there was positive correlation with increasing soil moisture (Ko and Lockwood 1968b). Kokke (1970a) found DDT to accumulate heavily in nursery soil, which adsorbed almost 100 percent of the DDT applied, while ditch water retained less than one percent. It was thought that the occurrence of relatively many DDT-resistant organisms of soil compared with relatively few resistant water organisms, was due to this high affinity of DDT for soil as opposed to water (Kokke 1970b). Shin and co-workers (1970) found precipitation to be the major factor in removing DDT from water when 4 ppb DDT were present. In soil, adsorption was found to be increased by removal of ether- and alcohol-soluble fractions from mineral soil, presumably by removing competition for limited numbers of adsorption sites (Shin et al. 1970).

Treatment of algae and protozoa with one ppm of DDT resulted in accumulation of 99 to 964 ppm, with no metabolites and no adverse effects observed (Gregory et al. 1969). Of 100 samples of estuarine and oceanic films, 35 were able to convert DDT to TDE, DDNS, and DBH during a 30 day incubation period. All the active films were estuarine while films from open water could not transform DDT at all (Matsumura and Boush 1972). Freshwater diatoms were able to convert DDT to DDE (Miyazaki and Thorsteinson 1972). Mayfly nymphs were observed to convert 85 percent of DDT to DDE within three days, and glass shrimp (*Palaemonetes kadiakensis* Rathbun) converted DDT to DDD, DTMC, and DBP; neither organism had reached a plateau in its accumulation of DDT in three days, and glass shrimp continued to accumulate DDT to higher and higher quantities for seven days (Johnson et al. 1971). No conversion of DDE to PCB (polychlorinated biphenyl) occurred in a terrestrial-aquatic ecosystem (Metcalf et al. 1975).

In chickens, DDT is metabolized to numerous compounds including DDD, DDA, DDE, and the 3-hydroxy- and 3-methoxy- analogs of these metabolites (Feil et al. 1975). The metabolism of DDT in pigeons was examined by Bailey and his co-workers (1969a, 1969b). It was shown that DDT had a half-life of about 28 days in all tissues and was converted, by separate pathways, to DDE and DDD. DDD in turn had a half-life of 24 days and was converted to DDMU, but DDE had a half-life of approximately 250 days.

In mice and hamsters, DDA was the principal metabolite of DDT. Both species excreted DDT, and conjugated DDT and its metabolites with glycine or alanine before excreting them in their feces, and mice, but not hamsters, excreted DDE in their urine (Wallcave et al. 1974). In rats, the conversion of *p,p'*-DDT to *p,p'*-DDD was carried out by intestinal flora rather than by the rat's own enzymes (Mendel and Walton 1966). McKinley and Grice (1960) reported that rats also metabolized dicofol to DDE.

Photolytic--Ultraviolet radiation decomposed DDT, being less effective if the soil contained humus than if humus-free soil or pure DDT was exposed (Piasecki et al. 1970). A free-radical mechanism for photochemical decomposition of DDT on quartz was postulated, with 4,4'-dichlorobenzophenone, 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane and 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene as the chief products. The reaction was 80 percent complete in 48 hours and did not necessarily produce hydrogen chloride (Mosier et al. 1969). On silica gel thin layer chromatographic plates, some aromatic amines sensitized decomposition of DDT by sunlight (Ivie and Casida 1971).

Seven days' exposure of solid DDT to ultraviolet light of greater than 230 nm (quartz glass) or greater than 290 nm (pyrex glass) resulted in the decomposition of some DDT to CO_2 and HCl. Of the initial 94 mg DDT, 89 were unaltered and 12 mg CO_2 and two mg HCl were produced. Under the same conditions, 98 mg of DDE resulted in formation of ten mg CO_2 and eight mg HCl while 85 mg DDE remained unaltered. No chlorine gas was produced (Gaeb et al. 1975).

Light of greater than 290 nm wavelength degraded dicofol to dichlorobenzophenone (Archer 1974). When dicofol was adsorbed to almond hull meal at 10.2 ppm and exposed to UV light, 50 percent was lost with 157 hours and 11 percent of the adsorbed dicofol was transformed into 4,4'-dichlorobenzophenone (Archer 1970).

Chemical and physical--Diffusion of DDT on homoionic clays was associated with conversion of DDT to TDE; the degree of decomposition depended on the type of clay and the exchangeable cation present (De Dios Lopez-Gonzalez and Valenzuela-Calahorro 1970). At temperatures above its melting point, DDT in the presence of metal salts decomposed to the noninsecticidal dichloroethylene (Balaban and Sutcliffe 1945). DDT was partially decomposed by 9*N* sodium hydroxide and by 18*N* sulfuric acid (Kennedy *et al.* 1972b). Complete decomposition was achieved in liquid ammonia and metallic sodium and substitution of lithium for the sodium gave better than 99 percent degradation (Kennedy *et al.* 1972a). Less than 20 percent of DDT was degraded by treatment with potassium permanganate while alkaline decomposition was successful (Leigh 1969).

In the laboratory, up to 90 percent of soil-applied DDT could be decomposed by mildly acidic reduction with zinc. Bis(*p*-chlorophenyl)ethane and tetrakis(chlorophenyl)tetrachlorobutane were the products (Sweeney and Fischer 1970, Sweeney *et al.* 1974).

DDT could be destroyed by burning in oxygen at 900°C with the formation of carbon monoxide, carbon dioxide, chlorine gas and hydrogen chloride, as well as other unidentified substances (Kennedy *et al.* 1972a, 1972b). Complete combustion of pure DDT was actually achieved at 560°C, but DDT formulated as technical flakes required 850°C for complete combustion (Kennedy *et al.* 1969). DDT dissolved in kerosene could also be destroyed in a blue-flame oil burner, and the hydrogen chloride gas which was generated was neutralized by an alkaline water spray. Powdered DDT required the addition of a baffled refractory furnace and a water-cooled feeder system to inject the powder, which then vaporized and burned. Dust formulations ranging from ten to 90 percent burned successfully (Whaley *et al.* 1970, Lee *et al.* 1971). Cutkomp (1947) observed that water-dispersed DDT could be decomposed by heating it to above 90°C, but boiling appeared to result in different products than did autoclaving, and aged or aerated dispersions did not decompose. The products were not fully identified.

Cobalt-60 *gamma*-irradiation of five Mrads decomposed 75 percent of a five ppm solution of DDT in hexane (Lippold *et al.* 1969). Woods and Akhtar (1974) were able to dechlorinate DDT which had been dispersed on silica gel suspended in water by use of *gamma*-irradiation. The level of irradiation was lower than that required to sterilize sewage, and so was considered economically feasible, but complete dechlorination was achieved with some difficulty. No data were available on the degradation of dicofol by chemicals or heat, but the results should be very similar to those for DDT.

Inasmuch as none of the data suggest that DDT, dicofol, or DDD will decompose to naturally occurring substances in either soil or water, thermal or chemical degradation appear to be the only practical ways of disposing of these compounds.

Transport:

Within soil--The fate of 414 lbs/A (207 ppm) of DDT and 22.1 lbs/A (11.05 ppm) dicofol applied to an Oregon orchard between 1946 and 1967 is shown in Table 39 and 40 (Kiigemagi and Terriere 1972).

In a 20-year study of orchard soils, less than one percent of the foliage-applied DDT was found 30-36 in. deep in the soil; most remained in the upper six inches (Terriere *et al.* 1965). Similarly, after 24 years of exposure to DDT, vineyard soil retained 88 percent of the recovered DDT in the top three centimeters of soil, while the figures were 95 percent and 92 percent respectively after 12 and six years (Kuhr *et al.* 1972). In Houston black clay, with 33 inches of rain per year, 60-75 percent of the DDT remained in the top 12 inches of soil after ten years (Swoboda *et al.* 1971). After ten years, 30 percent of

Table 39. VERTICAL DISTRIBUTION OF DDT ANALOGS AND METABOLITES IN ORCHARD SOILS, 1970 (KIIGEMAGI AND TERRIERE 1972)

Soil level, inches	Pesticide concentration, ppm					
	p,p'-DDT	o,p'-DDT	p,p'-DDE	p,p'-TDE	Dicofol	DBP
			Hood River			
0 - 6	33.1	5.59	7.61	1.59	2.44	7.25
7 - 12	7.25	1.20	0.97	0.18	2.32	0.87
13 - 24	1.08	0.17	0.19	0.02	0.50	0.19
25 - 36	1.02	0.11	0.12	0.01	0.36	0.07

Table 40. CHANGES IN VERTICAL DISTRIBUTION OF TOTAL DDT, 1965 - 1970 (KIIGEMAGI AND TERRIERE 1972)

Soil level, inches	Change, µg/sample	Change, %[a]
	Hood River	
0 - 6	- 7129	- 31
7 - 12	+ 1996	+ 159
13 - 24	- 331	- 16
25 - 36	+ 861	+ 217
0 - 36	- 4595	- 18

[a] As percent of 1965 levels.

the DDT residues were found in the six-nine inch layer (Lichtenstein *et al.* 1971a). Balinov (1973) found 97 percent of DDT residues in the top 30 centimeters, with 79 percent in the top ten centimeters, after 15-20 years; also after 15 years, DDT was found to have leached to 60 centimeters in a light sandy soil, even though only traces were recovered at this depth (Voerman and Besemer 1970).

In sandy loam, Stewart and Chisholm (1971) concluded there had been little downward or lateral movement in 15 years. Comparing silt loam and muck soil, Lichtenstein (1958) found 84-96 percent of DDT in the top three inches in loam, and only 62-74 percent in muck; in the three to six inch layer, loam held 4-12 percent while muck held 19-29 percent.

Leaching of DDT was found to increase when soil was cultivated (Harris and Sans 1969), when soil was treated with urea (Ballard 1971) and when calcium-containing rather than acidic homoionic clays were present (Lopez-Gonzalez and Gonzalez-Gomez 1970). However, there was some evidence that leaching was independent of the rate of application, since the leachate from gravelly forest soil contained 5 ug DDT per cubic decimeter (Riekerk and Gessel 1968). After eight years of DDT application, DDT had leached to 30 centimeters. More important, although only 0.3 mg/kg DDT (0.3 ppm) were found in the soil at the top of a hill, 7.23 mg/kg DDT were found in the soil at the foot of the hill (Naishtein *et al.* 1967). It is therefore apparent that, albeit slowly, DDT moves through soil, in soil, and with soil.

Between soil and water--Despite the virtual insolubility of DDT in water, contamination of rivers has occurred repeatedly (Breidenbach *et al.* 1967). Contamination of groundwater below orchards was detected (Ferriere *et al.* 1965). Relatively little DDT reaches groundwater through vertical leaching, however. Johnston and co-workers (1967) estimated that drainage tiles contained only about 1/7 to 1/12 the DDT carried by surface runoff and Johnson and Morris (1971) found DDT was carried in runoff from fields into the rivers. Of the DDT entering surface waters from cotton plots, less than three percent was in the water itself and over 96 percent was associated with sediment (Bradley et al. 1972). It was estimated that, in sea water, the humic acid fraction of the sediments carried 50 percent of the adsorptive capacity and was the most important DDT repository (Pierce et al. 1974).

The predominant pattern of DDT contamination of aquatic environments consists of extremely low or undetectable levels in water with the levels in mud and in water organisms increased by one or more orders of magnitude. Even under conditions of minimal water contamination, fish and aquatic invertebrates were found to be contaminated with DDT (Kuhr et al. 1974, Moubry et al. 1968).

In the Everglades, DDT was present at less than 0.03 ug/liter (0.03 ppb) in water, but levels in the underlying marsh soil were of the order of 30 ppm, and DDT levels in fish, omnivorous crustaceans, and algal mats ranged to 100 ppm (Kolipinski et al. 1971). In a Long Island estuary, concentrations in water birds were approximately a million-fold greater than the water levels (Woodwell et al. 1967). In a drainage system of southwestern Ontario, concentrations of DDT and its metabolites in mud were 820- to 13,000-fold as great as water levels of DDT, while fish carried up to 80,000 times the levels found in water (Miles and Harris 1971). Other reports of DDT magnification in aquatic systems abound (e. g.: Ettinger and Mount 1967, Reed 1969, Reinert 1970, Fredeen and Duffy 1970).

Actual degradation of DDT, even to DDD and DDE, is quite slow in aquatic systems, with no degradation observed in bottled river water after eight weeks (Eichelberger and Lichtenberg 1971) and no visible degradation observed in a sewage lagoon (Halvorson et al. 1971). Albone and co-workers (1972) reported that conversion of DDT to DDD occurred most effectively in an anaerobic sludge at 35°C; with a hydrogen atmosphere. In this study, the relative degradation of DDT to DDD was found to be slowest *in situ* in the Severn estuary sediment (Albone et al. 1972).

In the Ogallala aquifer, most of the DDT introduced into the water remained close to the point of entry and was not recovered (Scalf et al. 1969). Adsorption of DDT by suspended sea water particles was found to be associated with humic acid fractions (Pierce et al. 1974), and to be related to the size of the clay particles and the organic content of the particles (Leland et al. 1973). Adsorption by kaolinite and montmorillonite was slow, and equilibrium was reached only after one month (Weil et al. 1972). Off the coast of southern California, DDT levels increased between 1949 and 1970 in ocean and in fish with the residues decreasing with distance from a DDT-dumping sewer. DDT levels reached a plateau before 1970, and after dumping of DDT ceased in 1970, DDT levels began to decrease. DDE levels, which had been increasing steadily throughout, continued to accumulate, as did DDD, until the study ended in 1973 (MacGregor 1974).

The extremely slow movement of DDT within soil or from soil to water merely delays aquatic accumulation. In agricultural areas draining into south Monterey Bay, agricultural use of DDT declined sharply beginning in 1972, but DDT levels in marine sediment were still increasing in 1973 (Phillips et al. 1975). It was estimated that concentrations in sediment had reached 0.1 percent of the agricultural level of 2 lbs/A (2.24 kg/ha) by 1973.

Volatilization--The loss of DDT from a placid aqueous surface was found to occur by codistillation of DDT with water, with the rate of loss paralleling increasing DDT concentrations at least to 100 ppb. Moreover, DDT was found to have an affinity for the air-water boundary resulting in heterogeneous dispersal of DDT in the aqueous medium in a manner facilitating codistillation (Acree et al. 1963). These results were confirmed for DDT in sand, but not in soils of high organic content (Bowman et al. 1965).

The rate of loss of DDT from fields was estimated to be 2 lbs/A per year (2.24 kg/ha/year) in the summer, and 0.3 lbs/A per year (0.34 kg/ha/year) in the winter, so that about 1/2 of the DDT applied to fields might be expected to volatilize (Lloyd-J 1971). Freed et al. (1972) calculated, however, that the theo loss of DDT from an inert surface would not exceed 0.16 lb/A (0.18 kg/ha) per year.

Cliath and Spencer (1972) found that 66 percent of the volatile substance over a field containing DDT residues was actually *p,p'*-DDE, which has a higher vapor pressure than DDT. At 30°C, the volatile products of spraying one kg/ha of DDT consisted of: 62 percent *p,p'*-DDT, 16 percent of *o,p'*-DDE, 14 percent *p,p'*-DDE and eight percent *o,p'*-DDT (Spencer and Cliath 1972).

Cliath and Spencer noted that, at 30°C, *p,p'*-DDE has over seven times the vapor pressure of *p,p'*-DDT. More recently, Ware, Cahill, and Estesen (1975) compared the volatilization of DDT and DDE and concluded that DDE volatilizes more readily from a cool moist soil than from a warm dry soil. On, but not in, dry soil, *o,p'*-DDT was converted to *o,p'*-DDE. Volatilization of *o,p'*-DDT was more rapid from dry soil than from wet soil, with conversion to DDE occurring after, as well as before, volatilization. On both dry and moist soil, *o,p'*-DDT volatilized more rapidly than *p,p'*-DDT (Ware et al. 1975).

When ^{14}C-DDT was applied to different soils at levels of ten ppm and the soil was carried through three cycles of drying and rewetting, fine soil was found to maintain DDT volatilization for the longest time, presumably by retaining the requisite surface monolayer of water longer. Volatilization above 15 bars of water tension depended on temperature and on the adsorptive capacity of the soil. Evaporation was greatest from loamy sand, followed by silty clay loam; clay permitted least volatilization of DDT (Guenzi and Beard 1970). Sundaram (1974) found only small changes in the DDE levels from ground level to six feet, and found that *o,p'*-DDT dissipated more quickly than did *p,p'*-DDT.

In the laboratory, DDT volatilized from Gila silt loam at a rate corresponding to five kg/ha/year, with volatilization increasing as air flow increased. The rate of volatilization decreased with increasing time as the concentration of DDT in the soil decreased (Farmer et al. 1972). Flooding was found to decrease the volatilization of DDT, at least in part by changing the conversion product to DDD, which has a lower vapor pressure than the aerobically generated DDE (Spencer et al. 1974). Over a 48 hour period, DDT volatilized from unflooded soil at an average rate of 100 ng/m^3 and from flooded soil at 38 ng/m^3. For DDD, volatilization was 92 ng/m^3 and 30 ng/m^3 from unflooded and flooded soil, respectively. As expected, high wind and low rainfall increased volatilization (Willis et al. 1971). Mackay and Wolkoff (1973) stressed that even compounds with a low vapor pressure may evaporate rapidly due to high activity coefficients which result in high equilibrium vapor partial pressures.

Other modes of pesticide entry into the atmosphere included spray drift as particles of less than five nm, transport with soil in wind erosion, and transport with smoke from incinerated items contaminated by DDT (Brooks 1974).

In the atmosphere, these losses of DDT to volatilization are reflected in the presence of 57×10^{-15}g/m^3 of p,p'-DDT and 9×10^{-15}g/m^3 p,p'-DDE over the northern equatorial Atlantic ocean (Prospero and Seba 1972). The pesticides were not dust-associated, and therefore were presumed not to be of relatively local African origin. In Arizona, six to 229 ppb of DDT were found in deer mice (*Peromyscus*) downwind from areas of DDT application and quail livers contained up to 2,800 ppb and soil residues ranged from 3.6-6,700 ppb (Laubscher et al. 1971).

Loss of insecticidal activity in sunlight was less for DDT than for dieldrin, chlordane, or methoxychlor. Formulation affected inactivation, with wettable powders least sensitive, fuel oil or emulsified preparations intermediate, and kerosene solutions most sensitive

(Ginsburg 1953). Aqueous suspensions maintained toxicity to houseflies longer than did emulsions (Chisholm *et al.* 1949).

When five ppm DDT were applied to soil in the laboratory, the effects of temperature and ultraviolet light were found to interact, with loss of DDT from soil reaching 24 percent and 50 percent in 50 days at 30^{0}C and 50^{0}C, respectively. Ultraviolet light of 300-400 nm added four to eight percent loss at 30^{0}C and seven to nine percent loss at 50^{0}C (Baker and Applegate 1970). Level of application of DDT also affected loss: at 20 ppm, loss at 30^{0}C was 22-27 percent with ultraviolet light adding three to eleven percent; at 50^{0}C, 20 ppm resulted in only 35-44 percent loss, but the addition of ultraviolet light increased the losses by up to 32 percent. Finally, the soil type interacted with the other factors: clay was most effective in retaining DDT, while silty clay loam and gravel loam did not differ significantly (Baker and Applegate, 1970). DDT applied to leaves is lost by first order kinetics and at the same rate regardless of the rate of application (Gunther *et al.* 1946).

In a recent review of the movement of DDT and its derivatives, Spencer (1975) concluded that these compounds will continue to enter the atmosphere for many years after the use of DDT has been completely discontinued.

Into organisms--In a terrestrial-aquatic ecosystem, DDT and its metabolites were present at 0.004 ppm in the water, 22.9 ppm in snails, 8.9 ppm in mosquitos, and 54.2 ppm in mosquitofish (*Gambusia*). For DDE, the levels were 0.008 ppm in water, 171.6 ppm in snails, 168.9 ppm in mosquitos, and 149.8 ppm in mosquitofish. These data illustrate the bio-accumulation of DDT and DDE when compared with the DDT analog, methoxychlor, which was present in water at 0.0016 ppm, in snails at 15.7 ppm, in mosquitos at 0.48 ppm, and in mosquitofish at only 0.33 ppm (Metcalf *et al.* 1971).

In a watershed treated with one lb/A (1.12 kg/ha) DDT in 1960 and 1963, DDTR (total DDT residues) in 1971 were between 0.031 and 17.94 ppm in mud (mostly in dead water or beaver ponds), 0.045 to 0.196 ppm in crayfish (*Cambarus bartoni*) and 0.4 to 0.9 ppm in brook trout (*Salvelinus fontinalis*). Of the mud residues, 35 to 60 percent consisted of DDD, 30 percent of DDT, and 25 percent of DDE (Dimond et al. 1974). Schulz et al. (1975), using data from a Hawaiian canal and drainage ditch, suggested that small fish absorbed more DDT from the water than from food, while large fish absorbed more DDT from their food.

Miles and Harris (1971) found extremely small DDT residues in the water of a stream and drainage system of southwestern Ontario, but fish had accumulated up to 80,000 times the water levels of DDT in their tissues. The sum of these data makes it abundantly clear that extremely low levels of water contamination by DDT are indicative of nothing more than the very low solubility of the substance, and do not preclude high levels of contamination in aquatic organisms.

DDT is taken up from soil into wheat seedlings, with greater absorption from sand than from clay (Nash 1968). The rate of uptake was considered minimal (Harris and Sans 1969b) and was less than the uptake of dieldrin, endrin, or heptachlor (Beall and Nash 1969). When ten ppm of DDT were applied to the soil, rye, wheat and barley were found to take up 0.014, 0.032 and 0.057 ppm, respectively (Beitz et al. 1970). Uptake into peanuts was lowest in the nuts themselves, while levels in both turnips and turnip greens were considered to be possibly objectionable (Young 1969). In the Agro Pontino region of Italy, 0.058 ppm of DDT in the soil resulted in residues of 0.134 ppm DDT in beet roots (Camoni et al. 1971). DDT accumulated in root crops (MacPhee et al. 1960) and in grain at 0.1 to 1.2 ppm (Popov and Donev 1970). Uptake was not significantly correlated with soil pH, cation exchange capacity or clay content, but was correlated inversely with organic matter (Beall and Nash 1969, Harris and Sans 1969b).

Heinisch (1973) found that 141 days after application of dicofol at an unspecified level, carrots contained from 0.15 to 0.6 ppm of dicofol; Grandolfo and co-workers (1967) found that treatment with dicofol left unacceptably high residues on lettuce under conditions which resulted in acceptably low or no DDT residues.

Uptake of DDT by foliage was found to be in the order: emulsifiable concentrates > dusts > wettable powders, with consequent inverse toxicity to bees (Johansen and Kleinschmidt 1972). Application of DDT to spinach and cabbage leaves resulted in some transfer of DDT, DDE, and DDD to stem, roots, and soil as ascertained 10 days and 14 weeks after treatment (Kimmer and Klein 1972). Metabolites included DDE and DDMU on the leaves, and DDE, DDMU, DDD, DDA, and a DDA-conjugate and DBH conjugate in the leaves. Most of the radioactivity remained in or on the foliage (Kimmer and Klein, 1972).

The accumulation of DDT and DDE in animal fat is well documented (Woodward *et al.* 1965, Morgan and Roan 1971), including even species presumably remote from direct exposure, such as arctic mammals (Tatton and Ruzicka 1967, Clausen *et al.* 1974). Beef cattle sprayed with 0.5 percent DDT accumulated DDT in their fat and excreted it in their milk (Claborn *et al.* 1960). Chickens fed 0.1 ppm DDT for 16 days accumulated residues in their tissues; feeding of ten to 15 ppm for five days resulted in egg residues as well as residues in the abdominal fat; DDE residues of 0.2 ppm persisted for 26 weeks (Stadelman *et al.* 1965).

Persistence:

The persistence of DDT and its metabolites in soil is summarized in Table 41 for those cases where the residues remaining after a specified time could be quantified. After a total of 103.6 lbs/A (116 kg/ha) had been applied to a California soil over a five year period, the soil residues after the 11th application were between ten and 15 ppm. Four years later, four to six ppm remained in the soil (Hermanson *et al.* 1971). In agricultural soils in Finland, the average level of

Table 41. DEGRADATION OF DDT UNDER FIELD CONDITIONS

% Residue	# of Years	Loss/year %	Conditions	Reference
22	24	3.25	maximum dispersal	Kuhr *et al.* 1972
40	20	3.00	foliar sprays	Terriere *et al.* 1956
10.6	15-22	4.47	foliar sprays	Balinov 1973
39	17	3.59	maximum retention	Nash and Woolson 1967
55	15	3.00	sandy loam	Stewart and Chisholm 1971
18	15	5.47	10 lbs/A	Lichtenstein *et al.* 1971a
24	15	5.07	100 lbs/A	Ibid.
33	12	5.58	maximum dispersal	Kuhr *et al.* 1972
29	11	6.45	10 lbs/A	Lichtenstein *et al.* 1971a
56	11	4.00	100 lbs/A	Ibid.
16	10	8.40	Houston black clay	Swoboda *et al.* 1971
50	9	5.56	podzol soil	Krechniak 1973
44	8	7.00	25,50 lbs/A; bioassay	Fleming and Maines 1953
56	6	7.33	25,50 lbs/A; bioassay	Ibid.
50	6	8.33	maximum dispersal	Kuhr *et al.* 1972
64	4	9.00	25,50 lbs/A; bioassay	Fleming and Maines 1953
79	3	7.00	25,50 lbs/A; bioassay	Ibid.
50	3	16.67	isotope labeled	Czaplicki 1969
90	2	5.00	25,50 lbs/A; bioassay	Fleming and Maines 1953
97	1	3.00	25,50 lbs/A; bioassay	Ibid.
50	1	50.00	Texas soil	Randolph *et al.* 1960
55	0.75	60.00	pots: alkaline soil	Chawla and Chopra 1967
62	0.75	50.67	pots: normal soil	Ibid
100	0.75	0	bioassay	Mulla 1960
50	0.50	100.00	bioassay	Luchev *et al.* 1971
100	0.50	0	standing, percolated moist soil	Yule 1967
50	4	12.5	California soil	Hermanson *et al.* 1971

DDT and its metabolites was 0.073 ppm, or about 21 percent of the amounts applied. Data on the time span and the amounts applied to the soil were not available (Rautapaa *et al*. 1972). Beran and Guth (1965) found DDT to be less persistent than lindane, but more persistent than aldrin or parathion.

Enormous variation exists in the residues of DDT observed in different studies. After 24 years of DDT treatment of vineyard soil, despite maximum weathering, runoff, and volatilization due to bare soil and repeated tilling, 22 percent of the DDT remained in the soil (Kuhr *et al*. 1972). Under conditions favoring maximum retention (including soil incorporation, minimum tillage, and extremely high levels of application) Nash and Woolson (1967) found that 38 percent of a single application of DDT remained in the soil after 17 years. At the other extreme, it was estimated that only 50 percent of the DDT applied to Texas soil remained after one year (Randolph *et al*. 1960), and that less than 16 percent remained in a Houston black clay after ten years (Swoboda *et al*. 1971). The latter losses were ascribed to volatilization by the authors, who noted that soil temperatures reached 60°C.

Alkaline soil (pH 9.5) was reportedly conducive to DDT loss (Chawla and Chopra 1967) but pH differences between 6.0 and 7.5 did not alter degradation rates (Fleming and Maines 1953).

After DDT had been applied to a light sandy soil for 15 years, samples were taken at four year intervals. No significant decrease in the total soil residues of o,p'-DDT or of p,p'-DDT occurred in the four years, even though the soil was dug up yearly and either plowed or planted (Voerman and Besemer 1975).

When DDT was applied at ten lbs/A (22 kg/ha), 29 percent remained in the soil after 11 years, but when 100 lbs/A (112 kg/ha) were applied, 56 percent remained after 11 years. After 15 years, the corresponding residues were 18 percent and 24 percent (Lichtenstein *et al*. 1971a). After 1.5 years, a ten-fold increase in the rate of application was found to have resulted in a two-fold increase in the rate of retention (Lichtenstein and Schulz 1959b). The observation that higher levels of DDT result in higher rates of persistence has been made repeatedly (Nash and Woolson 1967, Lichtenstein *et al.* 1960).

The effects of soil must be evaluated separately for chemical persistence and biological activity. Addition of organic matter was found to increase conversion of DDT to DDD (Farmer *et al.* 1974). Organic colloids adsorbed DDT and reduced its volatility, so that at 126°C only 66-83 percent of the DDT was lost when organic soil colloids were added, whereas 100 percent of the DDT was lost at 80°C when colloids were not added (Porter and Beard 1968). When midwestern soils were ranked with respect to the length of DDT persistence, DDT was found to persist longer in heavily organic muck soils than in loam; but climate also affected the results, since soils from Kansas retained DDT longer than similar soils from Ohio or Wisconsin (Lichtenstein *et al.* 1960). DDT was found to adsorb virtually instantaneously to the surface of illite or kaolinite, but more gradually into montmorillonite, which is an expanding clay with intralamellar adsorption sites. The adsorption capacities of the clays were not correlated with their ion exchange capacities or specific surface areas (Ju-Chang and Liao 1970). On homoionic clays, cation exchange capacity was found to affect the dehydrohalogenation of DDT, and dried bentonite was found to adsorb three times as much DDT as dried vermiculite (Lopez-Gonzalez and Valenzuela-Calahorro 1970). Clay catalyzed decomposition of DDT was also found to increase with decreasing pH and to decrease with increasing amounts of organic base (Fowkes *et al.* 1960).

In evaluating the edaphic aspects of pesticide disposal, it was found that combinations of pesticide formulations were degraded more readily than single formulations, although, no attempt was made to determine whether the carrier or the active ingredient was responsible for the increased CO_2 evolution (Stojanovic *et al.* 1972a).

Paulini (1957) found colloidal clays, whether lateritic or not, to be highly adsorptive of DDT and suggested that ferric oxide did not inactivate DDT but only served as an indicator of the laterization of the clay. Downs and co-workers (1951) however, found that soils of high iron or aluminum content actually catalyzed DDT decomposition, with ferric chloride accumulating in the soil. Inactivation was not thought to be due to adsorption or to alkalinity of the soil, although catalysis by iron was implicated because one molecule of iron was found to degrade about 160 molecules of DDT before (presumably) decomposition products blocked further degradation (Downs et al. 1951). Gunther and Tow (1966) observed that ferric-catalyzed DDT composition could be inhibited by picolinic acid and salicylaminoguanidine. Iron porphyrins were found to mediate conversion of DDT to DDD in cell-free systems (Zoro et al. 1974).

Adsorption by organic matter, while apparently increasing the stability of DDT in the soil by decreasing its volatility and its decomposition, also decreased its availability to plants and insects (Harris and Sans 1967). When bioassays were used to measure residues, DDT was found to be least persistent in soils with high levels of organic matter (Fleming and Maines 1953). In forest soils, 91 percent of the DDT was found in humic acid extracts and only nine percent in fulvic acid fractions (Ballard 1971).

In orchards treated with 22.1 lbs/A (11.05 ppm) of dicofol between 1966 and 1967, Kiigemagi and Terriere (1972) found 5.62 ppm dicofol in the top 36 inches of soil in 1970. As shown in Table 40, heavy residues of DDT from a total application of 414 lbs/A were also found. Some of the dicofol may be due to conversion of DDT to dicofol, accounting for residues of 50 percent of the total application of 30 years. No other data on soil persistence of dicofol were available.

These results confirm the absence of microbial degradation of DDT and underline its incredible persistence. Despite numerous attempts, no evidence exists that DDT is converted to naturally occurring molecules under any soil conditions.

Effects on Non-Target Species-

Microorganisms--The data on the effects of DDT on CO_2 evolution, O_2 evolution, ammonification, nitrate production, and the overall numbers of fungi are shown in Table 42. Tables 43 and 44 summarize the effects of DDT on specific fungi and on bacteria. Table 45 shows the few data available on the effects of the DDT analogs, DDD and dicofol, on microorganisms. Levels of application, number of days observed and conditions of culture are also included. The interpretations of the effects as increasing, decreasing, or not affecting microorganisms (+, -, and 0, respectively) are repeated from the references cited and no critical reevaluation of the data was attempted.

It is quite apparent from Table 42 that DDT does not, even at massive doses, alter CO_2 evolution very much, nor affect the overall numbers of bacteria and fungi. Oxygen evolution from algal culture decreased sharply even at low levels (0.01 ppm) of DDT. Fisher (1975) noted that DDT depresses the growth rate rather than the rate of photosynthesis in marine phytoplankton, and concluded that total marine photosynthesis will probably not be diminished by DDT, even though selective toxicity could affect herbivore populations.

More fungi were stimulated than inhibited by DDT (Table 44), and the few inhibitions occurred at low levels, suggesting that DDT actually stimulated fungal growth, albeit only at massive doses. Bacteria, on the other hand, were more often inhibited than stimulated (Table 43) but applications of about 1,000 ppm (560 kg/ha) were required for inhibition.

Franzke and co-workers (1976) found that fungal stimulation was actually due to a combination of inhibition by DDT and stimulation by DDD; the observed affect consisted of the sum of these opposing effects.

Table 42. THE EFFECTS OF DDT ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	Level PPM	# days observed	Conditions	Reference
CO_2 evolution	0	2.24 kg/ha	1	30	pasture soil	Tate 1974
	-	280 kg/ha	126	30	pasture soil	Ibid.
	0		1,000		clay adobe soil	Bollen et al. 1954
	0		150-2,500		sandy loam soil	Bartha et al. 1967
	0				soil	Salonius 1972
O_2 evolution	-		0.01,0.1,1.0		algal culture: D. euchlora	Derby and Ruber 1970
	-		0.1,1.0		algal culture: P. tricornutum	Ibid.
Ammonification	-	.012-0.12	100-1,000	1,000	sandy black loam	Jones 1956
	0	.0012	10		sandy black loam	Ibid.
	0	50-200 kg/ha	22.5-90		soil	Tewari and Chakravarty 1970
	0	agr. levels	<0.5-2			Fearl 1952
	0		7.5,100,500		pasture soil	Ross 1974
Nitrate production	+	50-100 lb/A	25-50			Pathak et al. 1961
	0	0.05-2.0%	500-20,000			Wilson and Choudhri 1946
# of fungi	0	5-10 lb/A	5 x 5	>300	California soil	Martin et al. 1959
	+	50-100 lb/A	25-50			Pathak et al. 1961
	+ - -		1,000	>5	agar	Roberts and Bollen 1955
	0		1,10		agar	Ibid.
	0	5,50,500 mg/kg	5,50,500	30		[illegible] et al. 1971

+ = stimulation
0 = no effect
- = inhibition

Note: Estimate of effect as stated in the reference cited.

Table 42. THE EFFECTS OF DDT ON BACTERIA

Parameter	Effect	Level		# days observed	Conditions	Reference
		Original	PPM			
# of bacteria	0	20-25 kg/ha	9-11.3	1,3,10	light chestnut soil	Askerov 1969
	–	>40 kg/ha	>24	1,3,10	light chestnut soil	Ibid.
	0	20 lb/A	10	300		Martin et al. 1959
	–		7		agar	Ko and Lockwood 1968a
	0	agr. levels	<0.5-2		soil	Jones 1952
	0				soil	Salonius 1972
Nitrifying	–	0.15	1,000	<365	soil	Jones 1952
	0	20 lb/A	10	<300	California soil	Martin et al. 1959
	0	agr. levels	<0.5-2.0			Pant 1952
Nitrogen fixing	0	5,50,500 mg/kg	5,50,500	30		Drut et al. 1971
Spore forming	0	5,50,500 mg/kg	5,50,500	30		Ibid.
Ammonifying	–	0.15	1,000	<365		Jones 1952
Sulfur oxidizing	–	0.15	1,000	<365		Ibid.
[illegible]	0					Lemire and Prudette 1961
[illegible]	–					Ibid.
[illegible]	–				Laboratory	Trudgill and Widdus 1970
[illegible]	–					Lemire and Prudette 1961
[illegible]	–					Drut et al. 1971
[illegible]	0	5,50,500 mg/kg	5,50,500	30		
[illegible]	–	0.01 ppm	0.01		Laboratory	Cramer et al. 1974

Note: Estimate of effect as stated in the reference cited.
+ = stimulation
0 = no effect
– = inhibition

Table 16. THE EFFECTS OF DDT ON FUNGI

Parameter	Effect	Level		# days observed	Conditions	Reference
		Original	ppm			
Fusarium oxysporum	–		0.1		culture	Fromme et al. 1970
[illegible]	–	0.1-40 μg/ml	0.1-40		aquatic culture	Dalton et al. 1970
Aquatic hyphomycetes	–	≤1 μg/ml	≤1		aquatic culture	Ibid.
Microflora	0	1-1.1 kg/ha	0.25-1.1	7,3,17	light chestnut soil	Askarova 1969
	–	12 kg/ha	~1.9	7,3,10	light chestnut soil	Ibid.
	–	<1 (h)	<1		laboratory	Ries 1967
	–	1-20	1-20		laboratory	Ibid.
Phycomycetes	–	1-40 μg/ml	1-40		laboratory medium	Hodkinson and Dalton 1973
Hyphomycetes: aquatic	–	1-40 μg/ml	1-40		laboratory medium	Ibid.
terrestrial	–	1-40 μg/ml	1-40		laboratory medium	Ibid.
Actinomycetes	0	5,50,500 mg/kg	5,50,500	30		Dent et al. 1971
	–		10		agar	Ko and Lockwood 1968

Note: Estimate of effect as stated in the reference cited.

+ = stimulation
0 = no effect
– = inhibition

Table 45. THE EFFECTS OF THE DDT ANALOGS, DDD, AND DICOFOL ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	Level PPM	# days observed	Conditions	Reference
DDD						
CO_2 evolution	0		150-1,500		sandy loam	Bartha *et al.* 1967
Actinomycetes	–		1		agar	Ko and Lockwood 1968a
[illegible]	+			4	division in culture	Francke *et al.* 1970
Dicofol						
Azotobacter vinelandii	+	1.3 g/10 l	130			Peeters *et al.* 1975

Note: Estimate of effect as stated in the reference cited.

Increase = +
no effect = 0
Decrease = –

DDE was found to be neither stimulating nor inhibiting. Ko and Lockwood (1968b) found DDD to be more antimicrobial than DDT, with only one part per million of DDD required to inhibit actinomycetes, whereas ten parts per million of DDT were required. In soil, higher concentrations were required than in soil-agar plates. Parsenyuk and Akimenko (1970) found DDT to favor the denitrifying microflora because it inhibited the growth of nitrifying bacteria.

Despite these effects of DDT, the early reports of Smith and Wenzel (1947) and of Wilson and Choudhri (1946) that DDT does not deleteriously affect soil microorganisms have been reconfirmed. Vashkov (1969) found that a small bactericidal effect of combusted DDT aerosols on *Staphylococci* and on *E. coli* was due in part to the smoke. Trudgill and Widdus (1970) found DDT to have the least effect of seven insecticides on bacterial metabolism. It was postulated by Richardson and Miller (1960) that fungitoxicity is positively correlated with water solubility and vapor pressure and therefore DDT, with its extremely low solubility and low vapor pressure, exerted as little effect on fungi as the model predicted.

Dicofol applied at four times the normal agricultural levels did not decrease the yield of mushrooms (Poppe 1966).

Invertebrates--The effects of DDT on soil invertebrates have been exhaustively reviewed by Edwards and Thompson (1973) and need not be recapitulated here. DDT was not found to be mutagenic in either wasps (*Bracon hebeter*) nor in brine shrimp (*Artemia*), although extremely toxic to both organisms (Grosch and Valcovic 1967, Grosch 1967). At 0.05 ppm, DDT killed 90 percent of the larvae of oysters (*Crassostrea virginica*) and clams (*Venus mercenaria*) (Davis 1970).

Plants--DDT at 25 lbs/A (28 kg/ha) caused growth retardation in some seedlings, particularly cucurbitaceae, with some difference between technical and pure DDT (Cullinan 1949). The difference in purity was less significant than the effect of soils: DDT in mineral soil was more phytotoxic than in peat soil; the effects were considered to be due to decreased availability of phosphorus resulting curtailed lateral root development (Thurston 1953). Goldsworthy and Foster (1950) reported that the soil accumulation of 1,000 lbs/A (1,120 kg/ha) of DDT profoundly affected all plants, but that 3,000 lbs/A (3,360 kg/ha) on the soil surface did not damage the plants, presumably because there was no root contact. Dennis and Edwards (1964) found damage to tomatoes at all levels of DDT application with beans quite susceptible and cucumbers, carrots, and parsnips susceptible only at large doses. Damage was limited to potted plants, however, and little or no phytotoxicity was seen under field conditions. At 50 to 100 lbs/A (56 to 112 kg/ha), DDT delayed soybean emergence (Probst and Everly 1957). Boswell (1955) observed decreases in crop quality before either yield or growth were affected and more damage occurred in poor soil than in rich soil. Shaw and Robinson (1960) observed an unexplained increase in nitrification when DDT was applied at 20 and 200 lbs/A (22.4 and 224 kg/ha).

Fish and amphibians--The toxic effects of DDT on fish were pointed out by Pielou (1946), who noted that as little as 0.25 oz/A (.01 ppm) of DDT resulted in a complete kill of young fish unless weeds and mud were present. In the latter case, 3 oz/A (0.21 kg/ha) caused only a 70 percent kill (Pielou 1946). The acute oral LD_{50} for goldfish was found to be between 63 mg/kg and 200 mg/kg (Ellis *et al.* 1944), but the LC_{50} ranged from two ppb for largemouth bass and brown trout to 320 ppb for mosquito fish (Pimentel 1971). The effects of sublethal doses of DDT on fish included abortions in mosquitofish, changes in thermal acclimatization in salmon and trout, and loss of learned avoidance responses in trout (Pimentel 1971). The metabolism of DDT to DDD in trout was found to be enhanced by intestinal microflora, but was also carried out by liver enzymes (Wedemeyer 1968). DDT caused changes in schooling behavior of goldfish (*Carassius auratus*) when fish were exposed to one ppb for seven days and then transferred to clean water

(Weis and Weis [illegible]). The 48 hour LC_{50} for rainbow trout exposed to dicofol was 100 [illegible] (Pimentel 1971).

Frogs were killed by a single injection of 150 mg/kg DDT into the dorsal lymph sac (Ellis *et al.* 1944), but the LD_{50} for a single oral dose was greater than 2,000 mg/kg in bullfrogs (Tucker and Crabtree 1970). The relative toxicity of chlorinated hydrocarbons to anurans was estimated to be endrin > aldrin and dieldrin > DDT and toxaphene and resistance of up to 200-fold was found in amphibians from treated areas (Ferguson and Gilbert 1967).

Birds--The acute toxicity of DDT in birds ranged from an LD_{50} of 800 mg/kg in pigeons to an LD_{50} of greater than 2,200 mg/kg in mallards, while LC_{50} levels ranged from 300 ppm in bobwhites to 1,300 ppm in mallards when five day feedings were used as the criterion (Pimentel 1971). Tucker and Crabtree (1970) stressed the high degree of cumulative action of DDT as the effective minimum lethal dose in mallards was 50 mg/kg/day over a 30-day feeding period and 100 ppm over a one year period.

The LC_{50} for birds fed dicofol-treated feed for five days followed by clean feed for three days ranged from 1,400-1,500 ppm in two-week old coturnix quail to 2,800-3,000 ppm in bobwhites; mallards and pheasants were intermediate (Pimentel 1971).

At 500 ppm, dicofol in acetone injected into hens' eggs caused 30 percent mortality of the embryos (Dunachie and Fletcher 1969).

When pigeons were fed DDT, the observed increase in liver weight was more closely correlated with DDE levels than with DDT levels, and was not observed when DDD, which is not converted to DDE, was fed (Bailey *et al.* 1969a). DDE was toxic in the pigeon but did not cause the typical symptoms of organochlorine poisoning (Bailey *et al.* 1969b).

The drastic effects of chronic intake of DDT, and other chlorinated hydrocarbons, on avian reproduction have been adequately reviewed

(Pimentel 1971, Menzie 1972, Edwards 1973, Stickel 1971, Ware 1975, Peakall 1975). Haegele and Tucker (1974) fed 15 common pollutants to mallards and coturnix quail and only DDE caused eggshell thinning of prolonged duration after a single dose. Eggshell thinning was also the result of toxic effects by other compounds, but was rapidly reversed when their other symptoms waned.

Mammals--The acute oral LD_{50} of DDT in rats is 113 mg/kg (Servintuna 1963, Martin 1968, Brooks 1974) and the dermal LD_{50} is greater than 2,500 mg/kg (Martin 1968, Jones *et al.* 1968). The DDT metabolite and analog DDD (TDE) has an acute oral LD_{50} of 400 to 3,400 mg/kg and an acute dermal LD_{50} of greater than 5,000 mg/kg (Jones 1968). The acute oral LD_{50} for the DDT metabolites DDA and DDE is given as 740 mg/kg and 880 mg/kg respectively (Servintuna 1963). In the present array of insecticides DDT may be classified as moderately toxic to mammals if acute toxicity is the only criterion. Early evaluations generally concurred with the summary of Stammers and Whitefield (1947) that, properly used, DDT was harmless.

Toxicity in rats and rabbits was suggested to result from the presence of both aromatic and aliphatic chlorine, and to decrease with decreasing chlorination of the ethane bridge (Smith *et al.* 1946, Von Oettingen *et al.* 1946). Acute poisoning by DDT, other than by deliberate or accidental ingestion of large doses, is often allergenic, with allergic rhinitis, dermatitis, blood disorders, and autoantibody formation reported (Kagan *et al.* 1969). Therapeutic value was claimed for DDT by Greim (1970), who reported that in man, 90 mg DDT per day reduced hyperbilirubinemia by increasing bilirubin conjugation to glucuronic acid, and also found that two to five grams of DDD per day was beneficial in Cushing's syndrome since it decreased the excretion of 17-hydroxysteroids. A daily dietary intake of 2.2 mg/kg altered mast cell physiology and histamine-mediated reactions in rats (Gabliks *et al.* 1975). Hrdina and Singhal (1975) most recently reviewed the data pertaining to the pharmacological basis of DDT toxicity in mammals.

Accumulation of DDT in fat and its transmission into milk were noticed early (Woodward *et al.* 1945, Stammers and Whitfield 1947). Krasovskii and Shigan (1970) observed that rats fed less than 50 mg/kg/day accumulated DDT in their fat, but not in their livers, kidneys, brains, or hearts; compensatory increases in excretion were found as the dose of DDT was increased up to 50 mg/kg/day, suggesting that enzyme induction by DDT can modify the response to it. When rats were fed 10 to 20 mg technical DDT per day, serum and fat levels of DDT remained elevated for one year after feeding ended. Storage was found to be of the order: p,p'-DDE > p,p'-DDT > o,p'-DDT > o,p'-DDD (Morgan and Roan 1971). The conversion of p,p'-DDT to p,p'-DDD was found to be mediated by intestinal flora rather than by the rat's own enzymes (Mendel and Walton 1966).

In rats, DDT at 70 mg/kg/day increased liver phosphofructokinase and phosphofructoaldolase but inhibited glucose-6-phosphatase, glucose-6-phosphate dehydrogenase, and glucose-6-phosphate transketolase. All of these enzymes were inhibited by 1.5 mg/kg/day ([illegible] and Yakushko 1970). When 70 mg/kg/day were administered to rats, disturbances of serotonin metabolism were postulated because increases of 5-hydroxyindole-3-acetic acid were observed within two days. Chronic doses over three to five months somewhat reduced the excess excreted (Khaikina and Shilina 1971). Daily doses of one percent of the LD_{50} to rabbits rendered them more sensitive to coronospastic agents such as pituitrin (Kagan *et al.* 1974). DDT is generally recognized to be estrogenic (Cecil *et al.* 1971, 1975; Gellert *et al.* 1972, Nelson 1974). The DDT homologs, p,p'-DDD and DDE were not estrogenic, but DDA was somewhat estrogenic (Gellert *et al.* 1972).

In a lengthy review of the harmful effects of DDT, Kagan (1969) cited the effects of chronic intake of DDT in the rat to include marked alterations of liver functions at "large" doses (0.05 LD_{50} per day, or about 5 mg/kg/day). Moderate doses (0.01 LD_{50} per day, or about 1.2 mg/kg/day) resulted in decreased sleep, changes in blood serum enzymes, and increases in the weights of liver, kidneys, and heart. Small doses (0.001 to 0.005 LD_{50}, or about 0.1 to 0.05 mg/kg/day) caused minor changes in liver function and weight. It was stated that, in man, the effects of chronic DDT intake included higher rates of liver disease, central nervous system disorders, more endocrine disturbances, complications of pregnancy, spontaneous abortions, and premature births (Kagan et al. 1969). Radomski et al. (1968) observed increased levels of *p,p'*-DDE in patients dying of portal cirrhosis, cerebral hemorrhage, and hypertension.

Interactions of DDT with other pesticides included sharp increases in DDT storage in beagles when aldrin was added to a fixed regimen of DDT intake (Deichmann et al. 1971a), decreases in the toxicity of carbaryl when mice were pretreated with DDT (Meksongsee et al. 1967) and increases in dieldrin metabolism in DDT-treated female rats (Street and Chadwick 1967). Wagstaff and Street (1971) reported that guinea pigs, unlike beagles, stored less DDT when also treated with dieldrin. Synergism of toxic effects was reported between endrin and DDT in mice (Keplinger and Deichmann 1967).

The acute oral LD_{50} value of dicofol for rats has been estimated to be 1,100 mg/kg (Gaines 1969), 1,495 mg/kg, with 95 percent confidence limits of 1,039 to 2,150 mg/kg (Brown et al. 1969), and 575 to 2,000 mg/kg (Jones et al. 1968). For intraperitoneal injections the LD_{50} was 1,150 mg/kg, with 95 percent confidence limits of 867 to 1,525 mg/kg (Brown et al. 1969) or 17 mg/ml of a 20 percent solution (Gul'ko 1968). The acute oral LD_{50} of dicofol for rabbits was calculated to be 1,810 ± 350 mg/kg; for dogs, 4,000 mg/kg (Smith et al. 1959).

Chronic feeding of 1,250 ppm dicofol caused deaths in 3 months in rats (Smith et al. 1959) and 3,000 ppm caused deaths in five weeks (Verschuuren et al. 1973). Female rats were found to store more dicofol, and excrete it less rapidly, than males while growth was inhibited by chronic feeding of 100 ppm in females, but required 1,250 ppm in males (Smith *et al.* 1959). McKinley and Grice (1960) suggested that most dicofol would be stored as its metabolite, DDE.

After a single oral or intraperitoneal dose of dicofol, peak loads were observed in the heart and testes after 32 hours while in muscles, brain, and most other organs the peak occurred after 40 hours. In fat and feces the peak occurred after more than seven days (Brown *et al.* 1969). Non-lethal effects of dicofol parallel those of DDT, notably liver lesions and an increased liver weight relative to body weight (Smith *et al.* 1959). Dicofol, like DDT, increased the epoxidation of aldrin and of heptachlor in male rats and female quail, but it was less active than DDT in this respect (Gillett *et al.* 1966).

DDT does not appear to be teratogenic (Deichmann *et al.* 1971a, Schmidt 1973) but did cause reproductive disturbances in mink (Pimentel 1971) and racoons (Menzie 1972) at environmental levels. In beagles, 12 mg/day of DDT for fourteen months adversely affected the male libido, the female estrus cycle, pregnancy and milk production; stillbirths increased and litter survival decreased. Blood and fat levels in the adult beagles were comparable to levels found in human occupational exposure (Deichmann *et al.* 1971b). DDT was found to cross the placenta and was stored in fetuses (Bäckström *et al.* 1965, Schmidt 1973). When DDT or its analogs, methoxychlor, DDE or DDA, were administered to two, three, or four day old female rats, the rats remained anovulatory and showed persistent vaginal estrus when mature; male rats treated the same way were reproductively normal (Gellert *et al.* 1974).

Ozburn and Morrison (1964) were able to select for DDT resistance in mice, and after nine generations the selected line had an LD_{50} of greater than 900 mg/kg to DDT. This resistant line showed somewhat increased tolerance to dieldrin and lindane as well. In mouse lymphoma cells, resistance to DDT conferred resistance to dicofol, DDD, methoxychlor, and DDE. The resistance to dicofol was enhanced five times as much as DDT resistance. The resistance to DDT analogs was not due to enhanced degradation (Spalding _et al._ 1971).

Epstein and Shafner (1968) concluded that DDT was not mutagenic in mice, but Epstein and Legator (1971) cited data showing that DDT was mutagenic in mice if given to males at levels of 50 to 70 mg/kg. These data were later published by Palmer _et al._ (1973), who concluded that DDT was marginally mutagenic. Clark (1974) also considered DDT to be weakly mutagenic by the dominant lethal test in mice. *In vitro*, ten mg/kg DDT caused chromosome breaks and exchange figures in a marsupial cell line (Epstein and Legator 1971).

Considerable controversy exists as to the carcinogenicity of DDT. Nakamura (1960) observed precancerous lung changes in rats sprayed with DDT for three months, and chromoleukemia increased in rats fed a high-fat, highly purified diet. In the latter study, the diet rather than the DDT was considered the major carcinogen (Kimbrough _et al._ 1964). Terracini (1967) found suggestions of weak carcinogenicity of DDT in rats, mice, and dogs, but considered the data to be equivocal. Kemeny and Tarjan (1966) observed an increase of tumors from 1.22 percent in controls to 5.41 percent in treated mice, and 3.51 percent of leukemias in treated mice compared with little or no leukemia in controls. This study spanned five generations, and mice were fed 2.8 to 3.0 ppm per day, corresponding to 0.002 or 0.001 of the murine LD_{50}. More recent evidence for carcinogenicity in mice was reported by Tomatis and co-workers (1972) in a preliminary report on a projected seven generation study. Both in the parental and F_1 generations, DDT-treated mice had larger liver tumors earlier and more frequently than control mice. Aflatoxins were not present in the food, as had been suggested by Jukes (1970) about an earlier study. Lung tumors were found significantly more often in four of six generations of inbred strain A mice treated with DDT (Shabad _et al._ 1973) and liver tumors which were transmittable, but did not metastasize, were found in inbred Balb/c mice fed 250 ppm DDT (Terracini *et al*. 1973). DDT was also co-carcinogenic with methylcholanthracene (Uchiyama *et al*. 1974). DDT decreased the induction of tumors by dimethylbenzanthracene in rats (Okey 1972) and reduced the carcinogenicity of dialkylnitrosamines in rat, hamster, and mouse tissues (Bartsch *et al*. 1975).

Brown (1972) observed no effects on murine reproduction, and no teratogenesis, at dicofol levels up to 225 ppm; in rats, the no effect level was 100 ppm, and no pathological differences between control and treated rats were observed in the fifth generation at this level.

Methoxychlor

Methoxychlor is the common name for 1,1,1-trichloro-2,2-bis-(*p*-methoxyphenyl) ethane, introduced in 1954 by J. R. Geigy AG, and by E. I. du Pont de Nemours and Company, Inc. under the trade name Marlate.

Methoxychlor, a nonsystemic contact and stomach insecticide with a range of effectiveness similar to that of DDT, is synthesized by the condensation of chloral and anisole in the presence of acidic catalyst. The pure *p,p'*-isomer forms colorless crystals melting at 89°C and the technical product consists of about 88 percent *p,p'*-methoxychlor, most of the rest being *o,p'*-methoxychlor. Brooks (1974, vol. 1) detailed alternative ways of synthesizing methoxychlor. The technical product is a grey, flaky powder with a melting point of 77°C which is soluble in water to 0.62 ppm (Kapoor et al. 1970), moderately soluble in ethanol and petroleum oils, and readily soluble in most aromatic solvents. Brooks (1974) listed the solubilities of methoxychlor in kerosene and dichlorobenzene as 20,000 ppm (2 g/100g) and 400,000 ppm (40 g/100g) solvent.

Degradation:

Biological--Under laboratory conditions in Phillipine soils methoxychlor was degraded within one month in flooded Casiguran soil, within two months in flooded Luisiana soil, and within three months in flooded Maahas soil, while some residues remained after three months in flooded Pila soil. Under simulated upland (aerobic) conditions, methoxychlor residues were found in all soils after three months (Castro and Yoshida 1971). Degradation of methoxychlor by planktonic algae (*Chlorella*, *Monoraphidium*, *Ankistrodesmus*, *Koliella*, *Carteria*, *Scenedesmus*, and *Nitzschia*) ranged from 26 to 80 percent. Products were not identified (Butler et al. 1975). *Aerobacter aerogenes* degraded methoxychlor to 1,1-dichloro-2,2-bis(*p*-methoxyphenyl) ethane in two of four anaerobic cultures; the latter compound was also recovered from one of five aerobic cultures (Mendel *et al.* 1966).

In the terrestrial-aquatic ecosystem, methoxychlor was significantly less persistent than DDT, because of the biodegradation of the aryl CH_3O groups. Whereas DDT was converted to DDE, which was heavily stored in animal tissues, only small amounts of the corresponding methoxychlor ethylene were stored by animals. The principal degradation pathway for methoxychlor was conversion to the mono- and dihydroxy derivatives, followed by conversion to polar conjugates (Metcalf *et al.* 1971, Kapoor *et al.* 1970, 1972). On the basis of ecosystem studies, methoxychlor was ranked as a moderately persistent insecticide which, although severely toxic to fish, was more readily degraded by them than DDT (Metcalf and Sanborn 1975). The degradation products of methoxychlor found in the water of the model ecosystem were the monohydroxyethane, dihydroxyethane, dihydroxyethylene, and unidentified polar metabolites (Metcalf *et al.* 1971).

In fish, methoxychlor was readily metabolized to 2-(*p*-methoxyphenyl)-2-(*p*-hydroxyphenyl)-1,1,1-trichloroethane and 2,2-bis-(*p*-hydroxyphenyl)-1,1,1-trichloroethane (Reinbold *et al.* 1971). Excretion of ^{14}C-methoxychlor metabolites occurred chiefly through the liver and bile in rats (Weikel 1957). In mice, methoxychlor was O-demethylated to phenolic products, the major metabolites being 2-(methoxyphenyl)-2-bis-(*p*-hydroxyphenyl)-1,1,1-trichloroethane 2,2-bis(*p*-hydroxyphenyl)-1,1,1-trichloroethane and its ethylene, and 4,4'-dihydroxybenzophenone (Kapoor *et al.* 1970).

Chemical and physical--In aqueous alkaline solution methoxychlor loses hydrogen chloride to form the diphenylethylene derivative (Crosby 1969).

Photolytic--Under UV light in water about 50 percent of an original methoxychlor concentration of 0.1 to 0.2 mg/liter decomposed to form methoxychlor-DDE (a/a) and *p*,*p*'-dimethoxybenzophenone. In the absence of air, only the DDE analog was formed. Degradation was 90 percent complete after one month (Paris and Lewis 1973). When milk containing methoxychlor was exposed to UV light at 220 and 330 nm, the photoproducts were methoxyphenol, methoxychlor-DDE (p,p'), 1,1,4,4-tetrakis (p-methoxyphenyl)-1,2,3-butatriene, and p,p'-dimethoxybenzophenone (Li and Bradley 1969).

Transport:

Within soil--Methoxychlor was found to migrate as much as 100 cm under conditions in which 95 to 97 percent of the residues remained in the top ten centimeters of soil (Chuchowska 1972).

Between soil and water--Treatment of the N. Saskatchewan river with 0.309 ppm of methoxychlor resulted in water residues of 0.02 to 0.1 ppm 21 to 22 km downstream (Fredeen et al. 1975).

Into organisms--*Aerobacter aerogenes* and *Bacillus subtilis* accumulated methoxychlor directly from water. Uptake was extremely rapid and the total adsorbed varied linearly with water levels of methoxychlor between 0.5 and 5.0 µg/liter. Bacterial levels reached 1,400 to 5,300 times the water levels, and 80 to 90 percent of the uptake occurred within the first hour (Johnson and Kennedy 1973). In a river treated with 0.309 ppm of methoxychlor, some uptake by fish occurred, but residues disappeared within 17 days (Fredeen et al. 1975). Cattle fed methoxychlor-sprayed hay for six months, or fed up to six gm per day for six months, had no methoxychlor residues in their milk. At higher doses than six gm, 0.48 percent of the intake was excreted in the milk (Ely et al. 1953). Normal use of cattle sprays resulted in fat storage of 2.8 ppm after one treatment and 7.4 ppm after six treatments. Milk residues were 0.2 ppm after 24 hours and persisted no more than 21 days (Claborn et al. 1960).

Persistence:

Chuchowska (1963) found that methoxychlor, applied to soil at a rate of 2 mg/100 gm soil (20 ppm) essentially disappeared in 20 to 26 weeks if soil moisture was ten percent, and in 30 to 38 weeks if soil moisture was decreased to three percent. Methoxychlor was less persistent than either toxaphene or parathion (Obuchowska 1972).

In water, methoxychlor declined from application levels of 1.5 mg/liter to 0.5 mg/liter after 98 to 112 days, and to 0.15 mg/liter after 155 to 169 days. No further decrease was observed between 169 and 460 days (Luczak 1969). Agricultural residues of chlorinated hydrocarbon insecticides in Utah Lake included methoxychlor at inlets, but not at the lake's outlet; carp (*Carpio cyprinus*) were found to have residues between 56 and 62 ppb of methoxychlor, which compared favorable with DDT residues at the level of parts per million (Bradshaw et al. 1972). The half-life of methoxychlor in streams was two to seven days if biological degradation occurred, but 200 days if hydrolysis was the primary mode of degradation (Bender and Eisele 1971).

Effects on Non-Target Species-

Microorganisms--Methoxychlor did not significantly affect the numbers of fungi, the evolution of carbon dioxide, or the rate of nitrification, when mixed with potting soil at 12.5, 50, or 100 ppm (Eno and Everett 1958). Nitrification was not inhibited by methoxychlor concentrations of 2,500 ppm (Jones 1956), and carbon dioxide evolution was not affected by 2,500 ppm methoxychlor applied to sandy loam under laboratory conditions (Bartha et al. 1967). In contrast, an 85 percent reduction in photosynthesis was observed when phytoplankton was exposed to one ppm methoxychlor for four hours (Pimentel 1971), while 20 mg/liter (20 ppm) killed protozoa immediately and algal filaments within five days (Cabejszek 1965). Exposure of *Chlorella pyrenoidosa* to 0.1 ppm methoxychlor for 164 hours decreased growth by 19 percent (Kricher et al. 1975).

Invertebrates--Methoxychlor was toxic to the aquatic annelids *Asellus aquaticus* and *Lumbriculus variegatus* at 0.001 and 0.44 mg/liter, respectively (Lakota 1974). *Daphnia magna* were killed by eight mg/liter of commercial methoxychlor within 24 hours.

Plants--Methoxychlor did not significantly decrease root or top growth in beans at 12.5, 50 and 100 ppm in pot culture (Eno and Everett 1958).

Fish--The 48 hour LC_{50} for rainbow trout (*Salmo gairdneri*) was 0.0072 ppm; for guppies (*Lebistes reticulatus*), the 96 hour LC_{50} was 0.12 ppm (Pimentel 1971). The toxicity of methoxychlor to trout and bluegills (*Lepomis macrochirus*) decreased with increasing temperatures, especially during the first 24 hours (Macek et al. 1969).

Fathead minnows (*Pimephales promelas*) exposed to methoxychlor for 96 hours in continuous flow bioassays had a TL_{50} of 8.63 ug/liter, but hatching of eggs was inhibited at levels of one ug/liter and completely prevented by two ug/liter. In the same study all yellow perch (*Perca flavescens*) were killed by ten ug/liter of methoxychlor, and their growth was retarded by as little as 0.625 ug/liter (Merna and Eisele 1973).

Birds--Methoxychlor is of low avian toxicity. The acute oral LD_{50} for young mallards was 2,000 mg/kg, while the LC_{50} was 5,000 ppm when birds were fed treated feed for five days followed by clean feed for three days. The LC_{50} for pheasants, bobwhites, and coturnix quail was greater than 5,000 ppm (Pimentel 1971).

In hens and chicks fed methoxychlor for 85 and 56 days, respectively, fat and skin storage was observed at all levels of methoxychlor treatment (i.e., 2,4,8,10,100 and 1,000 ppm) while the higher levels of treatment also caused organ storage of methoxychlor. Sixty-three days after the end of methoxychlor feeding, 0.05 ppm remained in the skin and fat. After three weeks of feeding chickens methoxychlor, egg residue levels were 0.4 percent of the dietary levels, decreasing to less than 0.5 ppm within three weeks after methoxychlor was removed from the diet. Carcass residues were six to eight times as high as egg residues (Lillie et al. 1973). Low levels (one to ten ppm) were not estrogenic in six week old chicks (Foster 1973). No adverse effects on growth, food utilization, weight, or mortality were noted at any level (Olney et al. 1962).

Chicks from eggs injected with up to 500 ppm of methoxychlor were no more affected by post-hatching starvation than untreated chicks, nor was the hatching rate lower in methoxychlor-treated chicks (Dunachie and Fletcher 1969).

Mammals--The acute oral LD_{50} of methoxychlor in rats is 6,000 mg/kg (Martin 1968). In dogs, levels of 2,000 to 4,000 mg/kg/day induced convulsions after five to eight weeks (Tegeris et al. 1966). Kunze et al. (1950) reported growth retardation in rats fed 500 ppm/day of methoxychlor, apparently due to the unpalatability of the diet. Storage of methoxychlor in the fat of the rats was moderate at 500 ppm, slight at 100 ppm, and not detectable at 25 ppm; moreover, all detectable residues disappeared from the fat within two weeks after feeding of methoxychlor was stopped (Kunze et al. *op. cit.*).

Methoxychlor has some estrogenic activity at high levels (Welch et al. 1969, Nelson 1974, Harris et al. 1974), and 1,000 ppm methoxychlor in rams' feed increased the number of dead or tailless sperm somewhat (Jackson et al. 1969).

Methoxychlor was not carcinogenic when mice were injected with a single dose of ten mg or treated weekly with 0.1 mg on the skin, but the authors considered the test insufficient (Hodge et al. 1966). Radomski et al. (1965) considered methoxychlor a carcinogen which caused liver tumors at 2,000 ppm in mice. No conclusive data on the carcinogenicity of methoxychlor were available (Vettorazzi 1975).

CYCLODIENE INSECTICIDES

Aldrin, Dieldrin, and Endrin

Aldrin is the common name for 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-*endo*-1,4-*exo*-5,8-dimethanonaphthalene, a nonsystemic insecticide used primarily against soil insects. In the U.S., the name aldrin refers to a technical product of at least 95 percent purity; in Canada, to the pure compound. In Britain the pure compound is known as HHDN. Produced by the Shell Oil Company, in 1948, aldrin is a white crystalline, odorless solid; stable to heat, alkali and metals with a melting point of 104 to 140.5°C, a vapor pressure of 2.31 x 10^{-5}mm mercury at 20°C, and a water solubility of 0.027 ppm. It is readily soluble in organic solvents such as ethanol, (50 mg/ml), carbon tetrachloride (1,030 mg/ml) and turpentine (1270 mg/ml). It is produced by condensing the dehydrochlorinated Diels-Adler adduct of cyclopentadiene and vinyl chloride, bicyclo(2,2,1)-2,5-heptadiene, with hexachlorocyclopentadiene (Martin 1968).

Aldrin may be oxidized (commercially) with peracetic or perbenzoic acid to yield dieldrin: 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-*endo*-*exo*-5,8-dimethano-naphthalene, a nonsystemic insecticide of high contact and stomach activity to most insects. In the U.S., the term dieldrin refers to a technical compound of at least 85 percent purity. In Canada, dieldrin is the pure chemical, which in Britain is designated HEOD.

Dieldrin is stable to alkali, mild acid, and light, and gives no reaction with a Grignard reagent (Martin 1968). It forms white, odorless crystals with a melting point of 175 to 176°C, has a vapor pressure of 1.78 x 10^{-7}mm mercury at 20°C, and a water solubility of 0.186 ppm (Wurster 1971). Its solubility is 40,000 ppm in ethanol, 480,000 ppm in carbon tetrachloride and 170,000 ppm in turpentine.

Endrin is the common name for 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-*endo*-1,4-*endo*-5,8-dimethano-naphthalene. It is made by the epoxidation of isodrin with peracetic or perbenzoic acid. Isodrin (1,3,4,5,6,7,8,8-octachloro-1,3,3a,4,7,7a-hexahydro-4,7-methano-isobenzofuran) is made by the slow reaction of cyclopentadiene with the condensation product of vinyl chloride and hexachlorocyclopentadiene, as described by Brooks (1974, vol. 1). Under the trade name Telodrin, isodrin was at one time marketed as an insecticide in its own right.

Endrin, a nonsystemic insecticide, is a white, crystalline solid which melts with decomposition above 200°C, has a vapor pressure of 2×10^{-7} mm mercury at 25°C. Its solubility in ethanol is 30,000 ppm, in carbon tetrachloride 510,000 ppm, and in turpentine 210,000 ppm.

Aldrin, dieldrin, and endrin are related in that endrin is isomeric with dieldrin, and dieldrin is the epoxide of aldrin. Isodrin, an intermediate in the formation of endrin, is isomeric with aldrin, and endrin is its epoxide.

Degradation:

Biological--Some degradation of cyclodiene insecticides by soil microorganisms is well documented, particularly the conversion of aldrin to dieldrin (Gannon and Bigger 1958, Tu *et al.* 1968, Decker and Bigger 1965). Experiments on degradation of cyclodienes in the laboratory are summarized in Table A6. It is noteworthy that only in one case was it claimed that one organism, *Trichoderma koningii*, converted even three percent of the dieldrin to carbon dioxide (Bixby *et al.* 1971). Of twenty cultures which were successful in partially degrading dieldrin, all were able to degrade endrin to keto-endrin, but only thirteen were able to degrade aldrin to dieldrin (Patil and Matsumura 1970). In a series of 150 soil cultures, twenty-five could convert endrin to keto-endrin (Matsumura *et al.* 1971); even more strikingly, only 'a few' of

Table 46. DEGRADATION OF ALDRIN, DIELDRIN, AND ENDRIN UNDER LABORATORY CONDITIONS

Parameter	Product	Reference
Aldrin		
Chlorella pyrenoidosa	*	Elsner et al. 1972
Penicillium funiculosum	dieldrin keto-aldrin	Murado-Garcia and Baluja-Marcus 1973
92 cultures	dieldrin; 7 metabolites	Tu et al. 1968
13/20 dieldrin-degrading organisms	6,7-dihydroxy-aldrin	Patil and Matsumura 1970
Dieldrin		
microbial cultures	photodieldrin; traces	Vockel and Korte 1974
Trichoderma viride	unidentified	Matsumura and Boush 1968
6 *Pseudomonas*; 2 *Trichoderma viride*		Matsumura and Boush 1967
soil, H_2O, cow rumen, rat gut	photodieldrin	Matsumura et al. 1970
Trichoderma koningii	CO_2	Bixby et al. 1971
Pseudomonas sp.	**	Matsumura et al. 1968
sewage lagoon	0	Halvorson et al. 1971
Endrin		
20/150	ketoendrin	Matsumura 1971
20/20 dieldrin degrading cultures	ketoendrin	Patil and Matsumura 1970

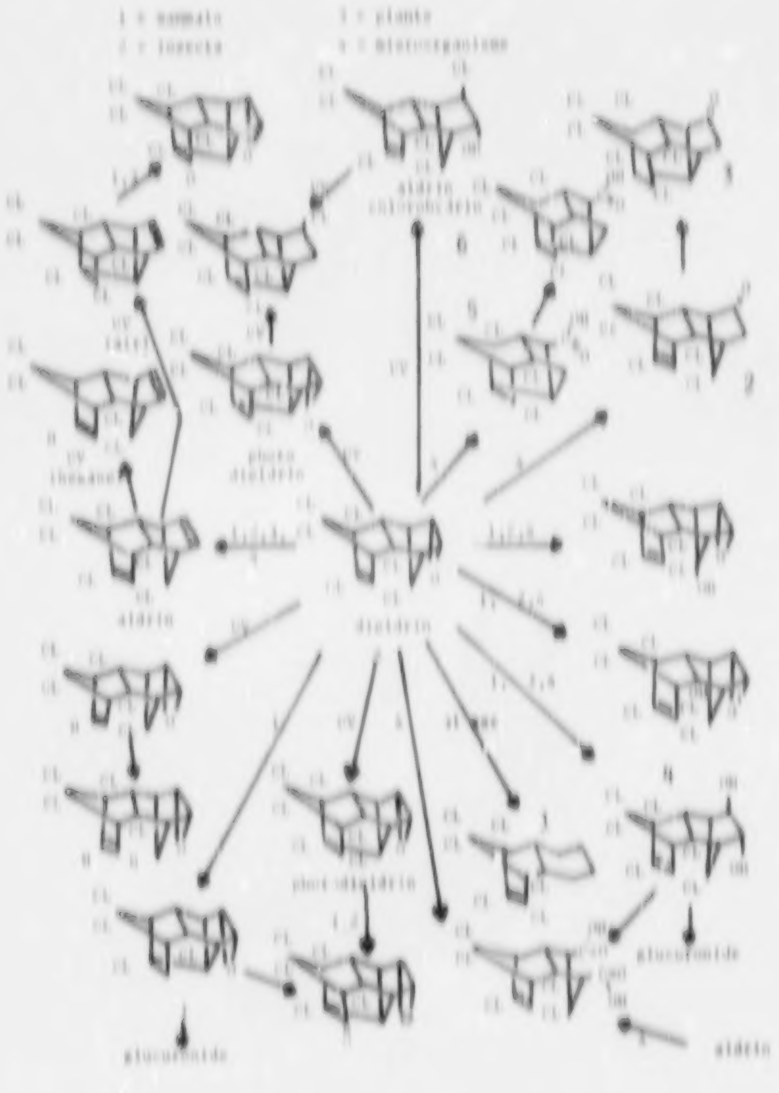
* Compound (1) in Figure 7

** Compounds (2,3,4,5,6) in Figure 7

500 cultures from heavily contaminated soil taken from the manufacturing site were able to degrade dieldrin; six of the successful cultures were *Pseudomonas* and two were *Trichoderma* (Matsumura and Boush 1967). For aldrin, the conversion to dieldrin is reportedly more effectively carried out by fungi and actinomycetes than by bacilli; after three weeks some further degradation, presumably of dieldrin, was observed in mixed cultures, but no metabolites were identified (Tu *et al*. 1968). Vockel and Korte (1974) reported photodieldrin as the only major metabolite of dieldrin, but recorded traces of unidentified metabolites also. Photodieldrin was also the predominant degradative product when dieldrin was incubated with soil, or with microorganisms from Lake Michigan water, from rat intestines, or from bovine rumens (Matsumura *et al*. 1970). Degradation products of aldrin and dieldrin are illustrated in Figure 8; those of isodrin and endrin in Figure 9.

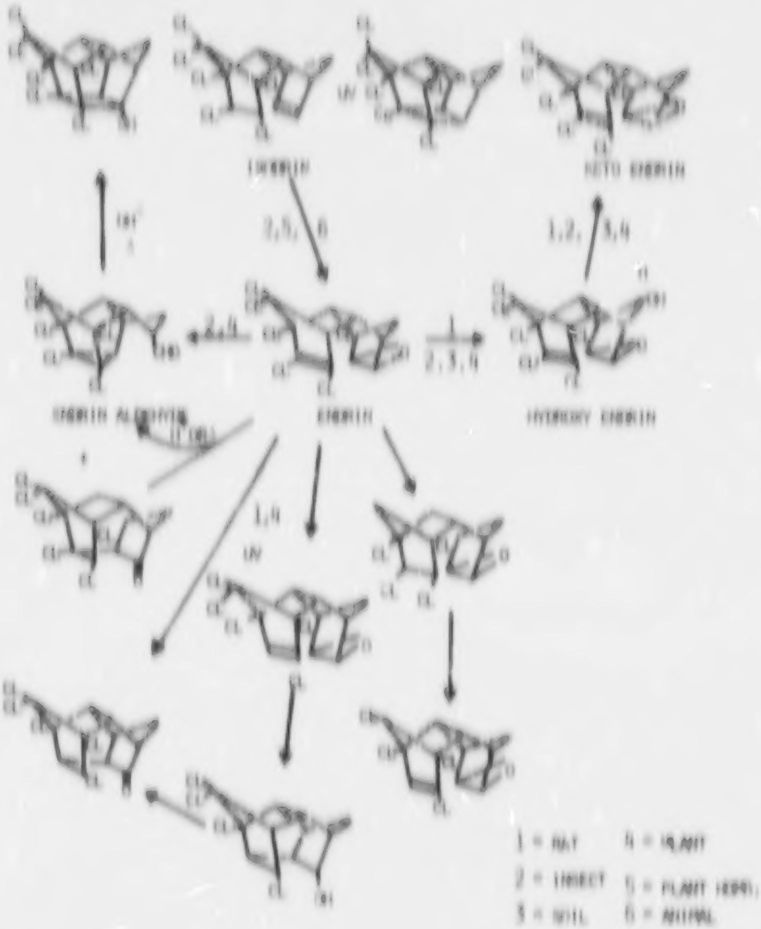
Despite the limited success of cyclodiene conversion in the laboratory, extrapolation to field conditions is difficult (Cowley and Lichtenstein 1970). No degradation of dieldrin in sewage lagoon sediment was observed (Halvorson *et al*. 1971). In simulated flooded and upland soil conditions, Castro and Yoshida (1971) found that aldrin disappeared more rapidly under flooded-soil conditions, but dieldrin proved persistent in both environments. When one hundred samples of estuarine and oceanic surface films, marine plankton, and algae were incubated with cyclodienes, only 35 of 100 estuarine cultures were able to degrade aldrin, dieldrin and endrin to *trans*-aldrindiol, photodieldrin, and keto-endrin, respectively, but none of the water samples from open ocean were even that active (Matsumura and Boush 1972). Moreover, sea bottom sediment was ineffective in degrading dieldrin, aldrin or endrin; algae were necessary for activity (Patil *et al*. 1972).

Most recently, microbial degradation of dieldrin in waste composting was attempted, with singular lack of success. At least 97.3 percent of the dieldrin remained unaltered after three weeks (Mueller and Korte 1975). When dieldrin was exposed to microbes from soil heavily contam-


1 = mammals
2 = insects
3 = plants
4 = microorganisms
aldrin chlorohydrin
photo dieldrin
photodieldrin
dieldrin
aldrin
UV
glucuronide
glucuronide
aldrin

METABOLIC PATHWAYS OF ALDRIN AND DIELDRIN

Figure 8


ISODRIN
KETO ENDRIN
UV
2,5, 6
1,2, 3,4
2,4
1
2,3,4
ENDRIN ALDEHYDE
ENDRIN
HYDROXY ENDRIN
1,4
UV
1 = RAT
4 = PLANT
2 = INSECT
5 = PLANT HOMO.
3 = SOIL
6 = ANIMAL

METABOLIC PATHWAYS OF ISODRIN AND ENDRIN

Figure 9

inated with pesticides, no degradation was observed, and aldrin degradation extended no further than the production of dihydroaldrin*trans*-diol and dihydrochlordene-1,3-dicarboxylic acid, plus traces of 4-keto-aldrin (Vockel and Korte 1974). Photodieldrin was also recalcitrant to degradation by algae (Reddy and Khan 1975).

Although readily stored and heavily accumulated in mammalian fat, with sixteen hundred-fold accumulations reported in man (Zatz 1972), cyclodiene insecticides are excreted and, to some extent, metabolized by mammals. An acute dose of endrin has a half-life of one to two days in rats if the dose was 16 ug/kg or 64 ug/kg, but a half-life of six days if 128 ug/kg were administered (Korte et al. 1970). In the same article, it was reported that if 200 ug of ^{14}C-endrin were injected as a split dose, the radioactivity was excreted in the form of metabolites, not of endrin. Male rats retained 5.2 percent of the radioactivity after 24 hours; females retained 12.1 percent. This sex difference persisted on a chronic dose of 0.4 ppm; steady state storage was achieved in both sexes after six days, but females stored twice as much endrin as males. Datta and co-workers (1965) reported more efficient excretion of dieldrin by male rats, with unidentified polar compounds being excreted in the urine; the males were again approximately twice as effective in eliminating the cyclodiene as were the females. In rabbits, dieldrin was metabolized to *trans*-6,7-dihydroxy-dehydro-aldrin, with a toxicity one-half to one-sixteenth that of dieldrin (Korte and Arent 1965).

Muller and co-workers (1975) observed that rats and primates oxidized dieldrin directly, producing 12-hydroxy-dieldrin; mice and rabbits opened the epoxide ring to give aldrin-4,5-*trans*-dihydrodiol. Since the mouse metabolized dieldrin more rapidly than either rats or rabbits, the authors speculated that this might be correlated with the hepatomas observed primarily in this species. Conversion of the aldrin and dieldrin metabolite to dihydrochlordene-dicarboxylic acid was quite rapid in rats and 44 percent of the administered ^{14}C was eliminated as metabolites within one week with dechlorination as the major pathway (Lay *et al.* 1975). The photoisomerization product of dieldrin, fed to rats at 10 to 30 ppm for 13 weeks, resulted in the identical urinary metabolites as dieldrin (Baldwin and Robinson 1975).

Photolytic--After one month's exposure to sunlight on glass, aldrin residues consisted of 2.6 percent aldrin, 4.6 percent dieldrin, and 93 percent of other photoproducts, of which two were highly insecticidal (Rosen and Southerland 1967). Through the pathways of photoisomerization and photooxidation, dieldrin was converted to photodieldrin; which is resistant to further transformation (Crosby and Moilanen 1974). In ultraviolet light, the conversion of dieldrin to photodieldrin was 50 percent completed in one hour and two unidentified compounds were also formed (Benson 1971). On silica gel chromatography plates, the photoisomerization of aldrin, dieldrin, and endrin was accelerated by many compounds with high triplet-energy values although no direct correlation was found between specific triplet-energy values and photosensitizing activity (Ivie and Casida 1971).

Ultraviolet light of greater than 230 nm was found to further degrade the aldrin metabolite, dihydrochloroindene-dicarboxylic acid, which is formed in rat intestines; and Gaeb and his co-workers (1974a) suggested that the degradation should also proceed in sunlight. Degradation of solid aldrin, dieldrin, and endrin occurred in a current of oxygen (Gaeb *et al.* 1974b). Endrin isomerized completely in seventeen days in hot sun (southern California in June) with formation of pentacyclic ketone, but no dechlorination occurred (Burton and Pollard 1971). Spontaneous degradation of endrin without dechlorination in the dark was reported by Barlow (1966). Gaeb and co-workers (1974c, 1975b) observed that aldrin adsorbed on silica gel surfaces was converted to different photoproducts than solid aldrin on glass. They suggested that the adsorption monolayer on silica gel permits greater access of oxygen to the pesticide and that decomposition on glass is a less realistic model than decomposition from adsorption, at least for soil insecticides. Photodieldrin was degraded to carbon dioxide and hydrogen chloride by ultraviolet light in the refrigerator, photodieldrin was converted to two unidentified metabolites, but in the freezer, it was stable for 45 days (Reddy and Khan 1975). Hartley (1968) suggested that volatilization of pesticides might result in their degradation in the highly photochemically active ionosphere, if the chemical had escaped to 50 meters and then escaped upward by eddy currents.

Chemical and physical--Dieldrin and heptachlor were not degraded by Grignard reagents of $LiAlH_4$, nor in a mixutre of melted potassium hydroxide and potassium nitrate at 230°C, nor in alkaline medium at high pressures; but some degradation--albeit without dechlorination--occurred in methanol and benzene at 13 KBar pressure and 140°C with an acid catalyst (Roemer-Maehler *et al.* 1973). Dieldrin was partially decomposed in 30 percent H_2O_2, but not by ethanolamine (Kennedy *et al.* 1972b) and complete degradation was achieved by metallic sodium or lithium in liquid ammonia (Kennedy *et al.* 1972a). Endrin was partially degraded by alkali, but not by chlorine gas or potassium permanganate (Leigh 1969).

Heating dieldrin to 230°C caused partial dechlorination and resulted in residues of chlorinated benzoic and phthalic acids (Stojanovic *et al.* 1972b). Heating to 900°C in the presence of air decomposed dieldrin to CO, CO_2, Cl_2, and HCl, with unidentified compounds accounting for only 9.4 percent of the gases (Kennedy *et al.* 1972b). The authors had demonstrated earlier that incineration of pesticides at 900 to 1000°C results in more nearly complete degradation of a large variety of herbicides and insecticides than does chemical degradation (Kennedy *et al.* 1969). Thus, even though data on the incineration of endrin and aldrin were not given, it is very plausible that their thermal degradation at 900°C also would be feasible and complete.

Photodieldrin was degraded to a number of unidentified products by γ-irradiation, but the authors did not consider the technique, which required seven megarads, to be a technologically feasible mode of degrading residues of these pesticides (Vollner and Korte 1974). Irradiation of aldrin, dieldrin, and heptachlor with 0.5 to 7.5 megarads of ^{60}Co-γ-radiation and/or 10 to 20 megarads of β-radiation produced numerous unidentified residues which, it was speculated, might be easily biodegradable (Caurvels *et al*. 1974).

Transport-

Within soil--Chlorinated hydrocarbon pesticides are considered to be immobile on a subirrigated column system (Harris 1969). Dieldrin was found to exhibit 'minimal' leaching when several soils were placed in columns or sloping troughs; this minimal leaching was, however, affected by soil type (Thompson *et al*. 1970). In irrigated fields in the United Arab Republic, light soils such as sandy and calcareous sandy loams were found to release pesticides more readily than clay or clay loams (Gawaad *et al*. 1971). Isodrin was distributed more evenly than aldrin, dieldrin, toxaphene or chlordane after application of 73 of 146 kg/ha in Congaree sandy loam (Nash and Woolson 1968). In a sloping field of sandy loam soil, residues decreased most rapidly at high points; the buildup followed plow lines, suggesting washoff rather than leaching, and was more striking for aldrin (dieldrin) than for heptachlor (Peach *et al*. 1973). In hot, dry soil, lateral movement did not exceed 15 cm, and the data were not consistent with diffusion of dieldrin in water (Cliath and Spencer 1971).

In soil boxes 60 cm x 60 cm, filled with loam soil, leaching of ^{14}C-aldrin could be detected after several weeks, although only about ten percent of the radioactive compounds had migrated vertically after three years (Moza *et al*. 1972). Of this, ten percent, or one percent of the total radioactivity, moved laterally and even upward in adjacent boxes (Kohli *et al*. 1973). Application of aldrin was equivalent to

2.9 kg/ha, or a total of 103 mg of ^{14}C-aldrin per box. One metabolite was identified as dihydrochloroindene-dicarboxylic acid (Moza *et al.* 1972). It was later established that this compound could be further degraded by UV light (Nash *et al.* 1974).

When aldrin was incorporated four to six inches into the soil at a rate of 20 and 200 lbs/A (22.4 and 224 kg/ha), up to twice as much pesticide was recovered in the lower as in the upper half of the plot, which sloped from 5 to 15 degrees (Lichtenstein 1958). At 1.5 and 2.5 lbs/A (1.68 and 2.80 kg/ha), soil-incorporated aldrin remained mostly in the top three inches of soil: 90 percent at one year and 72 to 80 percent after three years, with no residues detected in the six to nine inch layer (Lichtenstein *et al.* 1962). Cultivation resulted in a 76 to 82 percent decrease in residues recovered after seven to eleven years (Lichtenstein *et al.* 1971b); moreover, pesticides penetrated without cultivation (Harris and Sans 1969). Leaching to 60 cm in light sandy soil was reported by Voerman and Besemer (1970). In mineral soil, 14 percent of the pesticides were found at this depth (Harris and Sans 1969). Carter and Stringer (1970b) reported that aldrin, dieldrin, *gamma*-chlordane, and heptachlor penetrated to the same extent when applied as emulsified concentrates; penetration was greater in sandy soils than in clay soils, with the latter retaining 70 percent of the pesticides in the upper layer regardless of moisture content. Nash and Woolson (1968) reported that the relative mobilities of a series of chlorinated hydrocarbons in sandy loam were BHC (lindane) > isodrin > heptachlor > endrin > toxaphene > dieldrin > aldrin > dilan > chlordane. Eighty-five percent of the aldrin and dieldrin, but not of the isodrin, remained in the upper 23 cm of soil, with the greatest concentration from 7 to 23 cm. McLand (1967) stated that 90 percent of the residues recovered from 200 lbs/A (224 kg/ha) of soil-applied aldrin or dieldrin were in the top three inches (7.5 cm) of soil 17 months later. The sum of these data makes it clear that, despite their relative immobility, considerable movement of the cyclodiene insecticides eventually occurs under field conditions because of their unusually great persistence.

Between soil and water--When 22 to 45 kg/ha were applied to soil in strips not less than 3.6 meters from a pond, 0.3 ppm dieldrin was recovered from the bottom mud of the pond only if the dieldrin was not incorporated into the soil and vertical motion resulted in residues of no more than 1.1 ppm below 15 cm in the soil after 81 weeks (Edwards *et al*. 1970). Endrin was found in runoff at no more than 3 ppb, and was reported to reach ground water only if drought led to soil cracking and rain then carried endrin-carrying soil particles into the fissures; an interval of 72 hours was reported to halve the amount of endrin so transported, compared to rain 24 hours after endrin application (Willis and Hamilton 1973). Over a 41 month period little loss of dieldrin from soil was attributed to runoff (0.07 percent) or washoff (2.2 percent); volatilization accounted for 2.9 percent even though the highest levels of volatilization undoubtedly occurred before measurements were made (Caro and Taylor 1971). Barlow and Hadaway (1958) concluded that dieldrin did not evaporate until it had diffused evenly through soil blocks and had been adsorbed to the soil.

Despite their extremely low levels of movement into water, cyclodienes are measurable contaminants of canals (Bevenue *et al*. 1972: dieldrin), rivers (Young and Nicholson 1951: endrin; Lauer *et al*. 1966: endrin; Morris and Johnson 1971, Johnson and Morris 1971: dieldrin) and aquifers (Wells *et al*. 1970: dieldrin). Even when water contained no measurable amounts of cyclodienes, bottom sediment and bottom dwelling organisms often contained measurable residues (Moubry *et al*. 1968; Leland *et al*. 1973). Because of the hydrophobic nature of the cyclodienes, they were found to enter water adsorbed to soil, especially when organic matter was present (Goerlitz and Law 1974). Aldrin can be so thoroughly adsorbed to loam that no toxicity is observed in bioassays (Lichtenstein *et al*. 1967). Bevenue and co-workers (1972) found dieldrin to accumulate 6,000-fold in algae, 9,000 fold in sediment and 32,000-fold in fish as compared to water levels in canals. The water residues were in the range of parts per trillion, and fish contained about one ppm of dieldrin. A similar conclusion was reached by Reinert (1970). Persistence of pesticides in bottled river water subjected to natural and artificial light for eight weeks was found to be 20 percent for aldrin as aldrin, with the remaining 80 percent recovered as dieldrin; 100 percent for endrin, dieldrin, and DDT. In contrast, methyl parathion, parathion, malathion and carbaryl had totally decomposed (Eichelberger and Lichtenberg 1971).

In lakes, aldrin was removed from water by flocculent bacteria such as bacilli, flavobacteria, and protaminobacter, which adsorbed and concentrated particulates; the fate of the sedimented aldrin is not known (Pfister 1971, Leshniowsky *et al*. 1970). Adsorption by inorganic matter was found to be slow, with equilibrium on kaolinite or montmorillonite reached only after one month (Weil *et al*. 1972). On natural aquifer sands, adsorption of dieldrin and lindane was in the nanogram per liter range, was unaffected by temperature or pH, and was reversible (Boucher and Lee 1972). On montmorillonite, however, increasing the pH from six to ten decreased adsorption; changes of temperature between 10° and 30°C had no effect, and the addition of sodium chloride gave inconclusive results (Huang 1971). A single large scale marine contamination was reported after the sinking of a dieldrin-carrying ship. Waters near the wreck initially contained 40 ppb which decreased to less than 0.0005 ppb within five months. Mollusks were found to contain 13 parts per billion, fish 0.9 to 23 parts per million of dieldrin and no information on the routes of dispersal or the contamination of the sediment was given (Simal *et al*. 1971).

After application of approximately one ppb of dieldrin to a small slough in Canada, residues were undetectable in mud and water after ten months, but persisted in some vegetation and in aquatic invertebrates for almost two years. Final concentrations in invertebrates ranged from 1.8 to 44.6 ppb and concentration in successive trophic levels was not seen (Rosenberg 1975). In a terrestrial-aquatic ecosystem, aldrin was almost completely converted to dieldrin during the thirty-three day cycle; in *Gambusia*, only 0.5 percent of the ^{14}C was stored as aldrin. Three minor degradation products were found in the fish, namely, 9-hydroxydieldrin, 9-ketodieldrin, and an unknown thought to be *trans*-dihydroxydihydroaldrin; these compounds were also found when dieldrin was examined. Aldrin was found to have a Biodegradability Index (BI) of 0.00014 in *Gambusia* and of 0.0017 in the snail (Metcalf et al. 1973).

Results for dieldrin were very similar to those for aldrin, with a BI of 0.0018 in fish and 0.009 in snails. The Ecological Magnification (EM) was 5,957 and 11,149 for dieldrin in fish and in snails, respectively; the EM for aldrin was 3,140 for fish and 44,600 for snails (Metcalf et al. 1973, Metcalf and Sanborn 1975). The extreme resistance of dieldrin to degradation was apparent in the high degree of extractable radioactivity (Ave. 91 percent for the organisms) and the total amount of unchanged dieldrin in the organisms (88 percent of extractable radioactivity) after 33 days. In the water, dieldrin was present at concentrations of 0.002 ppm, accounting for 15 to 28 percent of the radioactivity (Sanborn and Yu 1973, Sanborn 1974).

Endrin in terrestrial-aquatic model ecosystem studies was found to be extremely toxic to the salt marsh caterpillar, and killed the *Daphnia* and mosquito larvae repeatedly after the water levels reached 0.06 ppm. *Gambusia* died within hours of being added to the aquarium when introduced on the 30th day of the experiment. The toxicity persisted for more than sixty days, delaying the termination of the experiment to sixty-six days, rather than the usual thirty-three days. This toxicity paralleled the endrin-induced fish kills in the Mississippi reported by Breidenbach et al. (1967) and Barthel et al. (1969). Endrin was, however, slightly less persistent than dieldrin; its BI was 0.009 in fish and 0.0124 in snails, and the EM values were 1335 in fish and 49,218 in snails (Metcalf *et al.* 1973).

In Lake Poinsett, a shallow, eutrophic lake in South Dakota, the levels of aldrin increased more rapidly in the higher trophic levels than did dieldrin levels (Hannon *et al.* 1970). In the alga, *Ankistrodesmus amalloides*, adsorption of aldrin was greater than that of photodieldrin (Neudorf and Khan 1975). A clear case of increasing accumulation at higher trophic levels was observed when homogenated clams (*Rangia cuneata*) were fed to crabs (*Callinectes sapidus*) which had been exposed to dieldrin. The clams contained 193 μg/kg (0.193 ppm) dieldrin and the crabs which ate the clams concentrated the dieldrin 3.9-fold in five days, and 4.7 to 6.8-fold in ten days (Petrocelli *et al.* 1975).

The substance of these data is that aquatic systems are no more efficient in the degradation of cyclodiene insecticides than is soil, and removal from water is into sediments or organisms, where accumulation rather than degradation can occur. More data are available for dieldrin than for endrin, although the latter has been responsible for fish kills (Young and Nicholson 1951, Lauer *et al.* 1966, Breidenbach *et al.* 1967).

Volatilization--The global transport of chlorinated hydrocarbons is well established (Risebrough *et al.* 1968, Wheatley 1973, Bidleman 1976), as is the global contamination by these compounds (Wheatley and Hardman 1965, Tarrant and Tatton 1968, Tatton and Ruzicka 1967). Estimates of the amount of pesticide volatilized from treated fields were 2.8 percent for dieldrin and 3.9 percent for heptachlor, as measured by fiberglass filters suspended over the field (Caro *et al.* 1971). Mulla (1960) found that 56 percent of the dieldrin disappeared in 24 hours from fields when the temperature was between 36 and 140°F (2.2-62.2°C), as assayed by bioactivity against gnats and mosquito larvae. A 50 percent loss of aldrin within 24 hours was also observed with sterile agar; no hydrophilic substances were found and volatilization was suggested as the mechanism of loss (Lichtenstein *et al.* 1968).

When ten ppm of dieldrin were applied to untreated, sprinkled and flooded soils, volatilization was seven percent, 18 percent and two percent respectively in five months (Willis *et al.* 1972) which led the authors to conclude that their data were inconsistent with the theory of codistillation of Acree *et al.* (1963). In the laboratory, dieldrin covered with soil volatilized more rapidly as the overlying soil bulk decreased, and volatilization increased with time as the dieldrin diffusion into the overlying soil neared the steady state. Volatility was proportional to the initial concentration of dieldrin in the soil and was limited by diffusion (Farmer *et al.* 1973). Dieldrin moved to the surface of the soil during periods of low moisture and relative humidity and then volatilized rapidly when the soil was remoistened (Spencer and McCliath 1973). Maximum volatilization of dieldrin occurred when the relative humidity was 100 percent (and soil water loss was zero), with the rate dependent on the dieldrin concentration. When the relative humidity was less than 100 percent, volatilization decreased with decreasing soil moisture, but was independent of the rate of water loss (Igue *et al.* 1972). Relative humidity did not, however, affect the volatilization of dieldrin or of aldrin from glass surfaces (Phillips 1971). The rate of volatilization of dieldrin from Gila silt loam at 20° or 30°C was estimated to be five kg/ha/year, compared with 302 kg/ha/year for lindane and 22 kg/ha/year for DDT. The rate of volatilization increased with increasing temperature and decreased with decreasing soil concentrations of dieldrin (Farmer *et al.* 1972).

Into organisms--Organochlorine insecticides are considered weakly systemic in plants, since uptake is measurable but not sufficiently great to be insecticidal (Finlayson and McCarthy 1973). In several soils known to be heavily contaminated with pesticides, traces of aldrin, dieldrin, and endrin were found to be absorbed in the order: carrots > radishes > turnips > onions (Harris and Sans 1967). Recovery of aldrin was less common than recovery of dieldrin or endrin, which were absorbed in the order: sugar beets > carrots > potatoes > sugar beet tops > corn, oats, and alfalfa (Harris and Sans 1969). Soybeans, grown fifteen years after pesticides were applied to Congaree sandy loam, contained residues of dieldrin, endrin, heptachlor epoxide, endrin transformation products, and pentachlorocyclohexanes but aldrin, isodrin, heptachlor, BHC, toxaphene and dilan were not identified (Nash and Harris 1973). Beall and Nash (1969) found the degree of translocation into crop seedlings to be: DDT < endrin < heptachlor, with endrin and heptachlor accumulating to some degree. In this study, uptake was not correlated with soil pH, cation exchange capacity, or clay content. Harris and Sans (1972) found that insecticidal activity and absorption by plants were positively correlated with organic content of the soil rather than with the applied concentration of dieldrin. Lichtenstein (1959) found that plant uptake of these pesticides from soils was in the order: sandy loam > silt loam > muck. Carbon added to the soil reduced the uptake of aldrin, dieldrin, and heptachlor by pea roots in sand and in loam (Lichtenstein *et al*. 1968). Inhibition of uptake of pesticides by carrots and potatoes was greater than 50 percent and persisted for four seasons when 2,000 ppm (0.2 percent) carbon was added to soil (Lichtenstein *et al*. 1971c). Radioactivity from ^{14}C-dieldrin was recovered from plants as dieldrin, photodieldrin, and hydrophilic metabolites (Kohli *et al*. 1973).

Microorganisms have been found to be effective accumulators of cyclodiene pesticides (Ko and Lockwood 1968a) and adsorption by dead fungi as well as by live fungi, streptomycetes and bacteria has been reported (Chacko and Lockwood 1967). Dead yeasts were found to adsorb more dieldrin than live yeasts (Voerman and Tammes 1969). Such adsorption by soil microorganisms is a logical consequence of the low water solubility and high lipid partitioning coefficient of the cyclodienes (Rosenberg 1975).

Dieldrin, aldrin, endrin, heptachlor and heptachlor epoxide are known to be taken up by higher plants from the soil, and in some cases to accumulate, particularly in root crops (Morley and Chiba 1965, Bigger 1966, Young 1969, Saha and Lee 1970, Popov and Donev 1970, Camoni et al. 1971). Aldrin is converted to dieldrin in or on plants (Gannon and Decker 1958). The translocation of isodrin, and of its conversion products endrin and keto-endrin, into the soil after leaf application has been documented for cabbage (Klein et al. 1972), but not for forage crops (Wheeler et al. 1967).

When ^{14}C-aldrin was applied to soil on which potatoes were grown, 60 percent of the radioactivity was recovered as metabolites, chiefly dieldrin and dihydrochlordene-^{14}C-dicarboxylic acid, (1,2,3,4,7,8-hexachloro-1,4,4a,6,7,7a-hexahydro-1,4-*endo*-methylene-indene-5,7-dicarboxylic acid). Aldrin was recovered in the potato peel and pulp and photodieldrin was in the potato haulm when potatoes were grown in England, but not when they were grown in Germany. Water which leached from the plots contained dihydrochlordene-dicarboxylic acid (Klein et al. 1973). Subsequent analysis of the fate of ^{14}C-aldrin in corn, wheat, and soils produced similar results in that the conversion products in plants and soils were qualitatively the same as in potatoes and soil. Aldrin residues decreased with increasing soil depth, and decreased, in corn, in the order roots, leaves, stems, cobs. Less than ten percent of aldrin or dieldrin leached below 40 cm in the most permeable soil and harvest residues in the grain did not exceed 0.01 ppm (Weisgerber et al. 1974b).

Persistence:

The extreme functional persistence of the cyclodiene insecticides is due to their highly stable ring structures and to the toxicity of the few transformation products that are formed. Thus the major terminal residues of aldrin is dieldrin (Gannon and Bigger 1958) which has approximately the same mammalian toxicity (Pimentel 1971) and approximately half the insecticidal activity (Mulla 1960a) of aldrin; the photolytic product of dieldrin, photodieldrin, is two to three times as toxic to insects, but almost five times as toxic to mammals as dieldrin (Brooks

1974, vol. 1). After five annual applications of aldrin, 95 percent of the aldrin had 'disappeared' in one year when measured as aldrin, but assays of aldrin and dieldrin combined showed no significant reduction in residues after six years (Korschgen 1971).

Persistence was found to be greater in muck than in loamy soil (Lichtenstein *et al.* 1960), and in brown forest soils than in loess-sandy soils (Homonnay-Csehi 1971). Dieldrin did not decompose during six months' exposure in percolated and standing moist soil (Yule *et al.* 1967) and was considered stable in hot climates (Atabaev *et al.* 1970). A summary of the data on the persistence of aldrin, dieldrin, endrin, and isodrin is given in Table 47. Freeman and co-workers (1975) analyzed the disappearance of soil-incorporated dieldrin and concluded that four years was too short a period to determine the kinetics of dieldrin disappearance. Linear regression led to a "best estimate" of 12.9 years for 95 percent dissipation, with an 80 percent probability that the true value lay between 10.0 and 20.5 years. The authors stressed that a ten year disappearance time is a minimum estimate and would have to be revised upwards sharply if first-order kinetics proved to be valid for dieldrin disappearance.

The extremely variable residue levels which were found are due to the multiplicity of factors which affect persistence; e.g., soil type, pH, moisture, temperature, organic matter content, clay content, mode of application and rate of application.

Under conditions predisposing to maximum persistence (thorough soil incorporation, minimum tillage, and up to 448 kg/ha) Nash and Woolson (1967) found residues of 31 percent technical dieldrin fifteen years after dieldrin application and 40 percent of the applied aldrin as aldrin and dieldrin. In comparing tilled to untilled fields, it was found that almost six times as much aldrin and dieldrin was recovered from the latter as from the former (Lichtenstein *et al.* 1971).

Table 47. RESIDUES AND RATE OF DEGRADATION OF ALDRIN, DIELDRIN, ISODRIN, AND ENDRIN UNDER FIELD CONDITIONS. ALDRIN WAS MEASURED AS DIELDRIN, AND ISODRIN AS ENDRIN

% Residue	# of Years	Loss/year %	Conditions	Reference
Aldrin and Dieldrin				
10	21	4.3	incorp. as termiticide	Bennet *et al*. 1974
23	16	4.8	sandy loam	Nash and Harris 1973
31	15	4.6	for max. persistence	Nash and Woolson 1967
5.8	15	6.3	silt loam	Lichtenstein *et al*. 1971a
5.0	13	7.3	soil incorporated	Freeman *et al*. 1975
11.2	8	11.1	bioassay	Wingo 1966
50	3	3.8	dieldrin	Hermanson *et al*. 1971
"all"	6	-	bioactivity; loam soil	Korschgen 1971
50	6	8.3	aldrin applied	Hermanson *et al*. 1971
5	1	95	aldrin residue only	Ibid.
3-19	4.5	19.8	various soils	Lichtenstein *et al*. 1960
8-10	4.5	18.2	turf	Lichtenstein and Polivka 1959
40	3	20	isotope--labeled	Czaplicki 1969
~40	3	20	sugar plantation	Stickley and Hitchcock 1977
~33	2	33.5	sugar plantation	Ibid.
55	2	22.5	incorporated granules	Lichtenstein *et al*. 1964
15	2	42.5	surface granules	Ibid.
40	2	30	incorporated emulsion	Ibid.
6.5	2	46.8	surface emulsion	Ibid.
65	1	35	incorporated granules	Ibid.
16	1	84	surface granules	Ibid.
45	1	55	incorporated emulsion	Ibid.
8	1	92	surface emulsion	Ibid.
25	1.7	42.9	northern Tanzania	Park and McKone 1966
50	1	50	Texas	Randolph *et al*. 1960
Isodrin and Endrin				
16	16	5.2	Isodrin applied	Nash and Harris 1973
39	16	3.8	Endrin applied	Ibid.

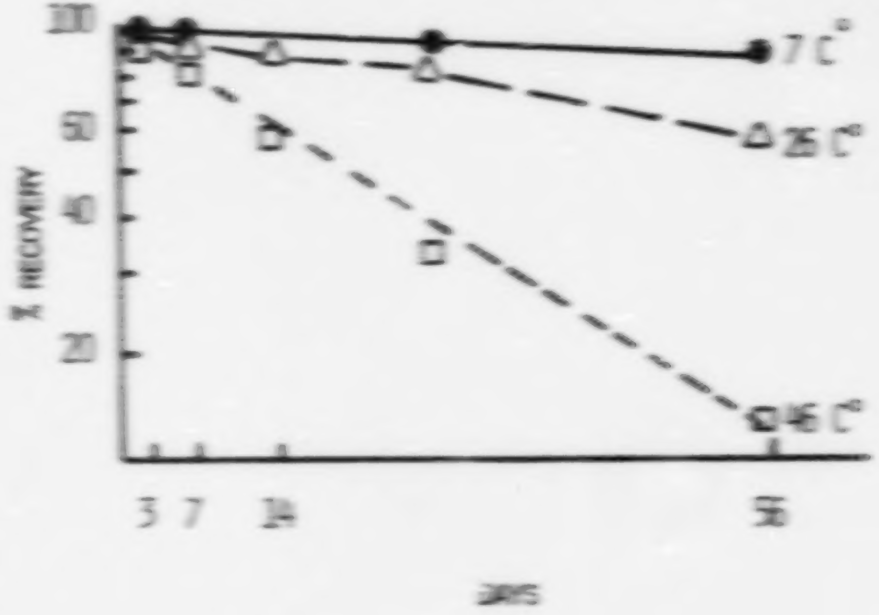
Data concerning the persistence of endrin are few. Mulla (1960b) found no loss of activity after nine months in soil. On air-dried soils at pH < 7, seven of ten endrin samples had isomerized after 48 hours; a ten to 80 percent loss had occurred within the first twelve hours (Asai *et al.* 1969). At pH of 6.4, ^{14}C-labeled endrin was transformed to a *delta*-ketone, an aldehyde, and an alcohol; the alcohol was not produced in moist soil at pH of 4.2, and none of the transformations took place in dry soil at pH of 4.2 (Nash *et al.* 1972). Endrin was found to be stable in alkali, and its transformation products were stable in all treatments (ibid.).

Lichtenstein and his co-workers (1964) applied aldrin as an emulsion and as granules, and each formulation was both surface applied and incorporated into the soil. The most persistent application was of granules incorporated into the soil, but formulation was of less importance than soil incorporation. After two years, soil incorporation resulted in residues of 55 percent for granules and 40 percent for emulsions, but surface application resulted in residues of only 15 percent and 6.5 percent for emulsions and granules respectively.

The type of soil affected both the biological activity and the persistence of cyclodienes in soil. In one study, it was found that the toxicity of dieldrin decreased linearly with increasing organic content in moist soil or with increasing clay content in dry soil; in mineral soil, toxicity increased with increasing moisture, but in muck soil, toxicity decreased with increasing moisture (Harris 1972). Lichtenstein and Schulz (1960) reported that the epoxidation of aldrin to dieldrin was more rapid in loam than in muck, with conversion 50 percent complete after three months in the laboratory at 30°C, and after 16 months in the field; moist soil resulted in more rapid epoxidation than did either dry soil, autoclaved soil, or sand. Increased organic matter was reported to decrease dissipation of dieldrin (Harris and Sans 1972). Recovery in chernozoic soil was reported to increase with increasing moisture (Head and McKercher 1971). It was postulated that the hydrophobic cyclodienes were adsorbed by soil particles in the absence of moisture, but competed for available adsorption sites in moist soils which, in the latter case, would result in increased toxicity and/or mobility (Harris 1964). It has been reported that dieldrin adsorbed almost instantaneously on kaolinite and illite surfaces, while it gradually diffused into the interlamellar spaces on the expanding montmorillonite clays; the adsorptive capacities of the clays for dieldrin were not correlated with either the ion exchange capacity or the specific surface areas of the clays (Ju-Chang and Liao 1970). Wiese (1964) found progressive biological inhibition as the clay content increased when organic matter content was held constant. Bollen and co-workers (1958) did not find any correlation between recovery rates of aldrin or dieldrin and either buffer capacity, ammonium binding capacity, cation exchange capacity, or pH. In another study, it was found that the nature of the clay minerals did not affect the amount of aldrin adsorbed, but the mechanical composition and organic content of the clays did. Organic matter added to the clays decreased the adsorption of aldrin (Yaron *et al*. 1967). Clays were also found to catalyze the decomposition of insecticides because acid diluents in the clay resulted in breakage of the epoxide rings of dieldrin and endrin, with formation of ketoendrin (Fowkes *et al*. 1960). Lower pH, higher temperatures, and lower levels of organic bases were found to increase the reaction rates.

Temperature also affected the amount and kind of terminal residues. It was reported that the epoxidation of aldrin to dieldrin did not occur at 7°C in loam (Lichtenstein and Schulz 1959a); dissipation from the soil in 56 days was found to be 16-27 percent at 6°C, but 86-98 percent at 46°C (Lichtenstein and Schulz 1959b). Effects of temperature on aldrin in soil are shown in Figure 10 (Lichtenstein and Schulz 1959b).

Finally, level of application was also found to affect the rate of loss of aldrin and dieldrin, with higher levels being conducive to slower



LOSS OF ALDRIN FROM A SILT LOAM AS AFFECTED BY TEMPERATURE (LICHTENSTEIN AND SCHULZ, 1959a)

Figure 10

dissipation (Nash and Woolson 1967, Wingo 1966). Data are shown for several chlorinated hydrocarbons in Figure 7 (Lichtenstein and Schulz 1959b).

Effects on Non-Target Species:

Microorganisms--The effects of aldrin, dieldrin, and endrin on soil microorganisms are summarized in Tables 48, 49 and 50. The variability of results, while possibly due to variations in treatment levels, methodology, and laboratory environments, probably also argues for great diversity of response by microorganisms. It has been shown that different marine phytoplankton genera may differ by 1,000-fold in their sensitivity to pesticides (Menzel *et al*. 1970); the same might be expected of soil organisms. Soils, moisture, and temperature variations between experiments may also have complicated comparisons, if only by affecting solubility or vapor pressure (Richardson and Miller 1960). The discordant effects shown in Tables 48, 49 and 50 augur well for soil response to even extremely high pesticide concentrations. Nevertheless, as Parr (1974) emphasized, the existence of even slight effects due to chlorinated hydrocarbons is serious because of their extreme persistence. Jones (1956) found that 0.01 percent of aldrin might reduce ammonification for more than three years; in paddy culture, insecticides were found to affect the availability of nitrogen, but not of phosphorus or potassium, for more than three months. The studies were usually of shorter duration, typically one to two months, and significant effects required the application of extremely large doses of pesticides.

Invertebrates--The effects of pesticides on soil invertebrates have been exhaustively reviewed (Edwards and Thompson 1973) and need not be detailed here. The authors concluded that the relationship between dose and effect tends to be logarithmic, minimizing chances of killing a large portion of the soil fauna; nevertheless, these pesticides commonly result in decreased species diversity and changes in predator-prey relationships, with previously harmless species occasionally increasing

Table 48. THE EFFECTS OF ALDRIN ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	Level PPM	# days observed	Soil or Condition	Reference
CO_2	+		200;1,000	10	10 Oregon	Fletcher and Bollen 1954
	-			18	sandy loam	Bartha *et al.* 1967
	-			30	" "	Ibid.
Ammonification	-		>100	>365	sandy loam	Jones 1956
	0	50 kg/ha	<22	3		Tewari and Chakravarty 1970
			1	3		Bardiya and Gaur 1968
Nitrate production	-		200;1,000	10	10 Oregon	Fletcher and Bollen 1954
	+		5,50	30	neutral alluvial	Brown 1954
	0		250	18	sandy loam	Bartha *et al.* 1967
Nitrite production	+		200;1,000		10 Oregon	Fletcher and Bollen 1954
# fungi	-	40 ug/ml	4,000			Cowley and Lichtenstein 1970
	+		100;10,000	>5	agar	Roberts and Bollen 1955
	+	50-200 lbs/A	25-50			Pathak *et al.* 1961
# bacteria (all)	+		200;1,000	10	10 Oregon	Fletcher and Bollen 1954
	0		4-220	60	clay, sand, peat	Jönsson and Fåhraeus 1960
	+	50-200 lbs/A	25-50			Pathak *et al.* 1961
(Nitrifying)	+	50-200 lbs/A	25-50			Ibid.
(Azotobacter)	0		200;1,000	10		Fletcher and Bollen 1954

+ = stimulation
0 = no effect
- = inhibition

Table 48. THE EFFECTS OF DIELDRIN ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Original	ppm	# days observed	Condition	Reference
CO_2 production	0		1	>3		Bardiya and Gaur 1968
	-		100-1,000	>3		Ibid.
Ammonification	-		>100	360		Jones 1956
	-	40-60 mg/kg	40-60	40	alluvial meadow	Rankov and Khristova 1971
Nitrate production	+	.005%	5	35		Brown 1954
	0	.005%	5	60		Ibid.
# fungi (all)	0	25,500 mg/kg	25,500	40	clay	Mahmoud *et al.* 1970
Actinomycetes	-	25,500 mg/kg	25,500	40	clay	Ibid.
	-	40-60 mg/kg	40-60	40	alluvial meadow	Rankov and Khristova 1971
Bacteria (all)	+→-	25,500 mg/kg	25,500	40	clay	Mahmoud *et al.* 1970
(Nitrifying)	-	40-60 mg/kg	40-60	40	alluvial meadow	Rankov and Khristova 1971

+ = stimulation
0 = no effect
- = inhibition
Change of effect with time shown by →

Table 58. THE EFFECTS OF ENDRIN ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level		# days observed	Reference
		Original	PPM		
CO_2 production	+	3 x seed dress.			Bollen and Tu 1971
	–	10 kg/ha	<22.2	7	Gawaad et al. 1972a, 1972b
	0	10 kg/ha	<22.2	15-45	Ibid.
Ammonification	0	3 x seed dress.			Bollen and Tu 1971
	+	10 kg/ha	<22.2	7	Gawaad et al. 1972a, 1972b
	+	10 kg/ha	<22.2	15-45	Ibid.
	0			40	Gawaad et al. 1973
Nitrate production	0	3 x seed dress.		7	Bollen and Tu 1971
	–	10 kg/ha	<22.2	15-45	Gawaad et al. 1972a, 1972b
Nitrate production	+	10 kg/ha	<22.2	15-45	Ibid.
# fungi (all)	–	10 kg/ha	<22.2	7	Gawaad et al. 1972a, 1972b
	0	10 kg/ha	<22.2	15-45	Ibid.
# bacteria (all)	–	10 kg/ha	<22.2	7	Ibid.

+ = stimulation
0 = no effect
– = inhibition

sufficiently to become pests. Subsequent literature has not changed these conclusions. Similar decreases in species diversity were reported for aquatic invertebrates (Wallace and Brady 1971). In a detailed assessment of the effects of 20 lbs/A of ten percent aldrin granules (2.24 kg/ha) applied to an Illinois stream in November of 1960, Moye and Luckman (1964) observed increases in caddis flies (*Trichoptera*) and midges (*Chironomidae*) through the summer following the aldrin treatment; mayflies (*Ephemoptera*) were reduced severely by the treatment and beetles (*Elmidae*) were apparently unaffected. The authors concluded that the effects were largely nullified by the second summer, at which time the numbers of caddis flies and midges had decreased to normal levels.

Davis and Hidu (1969) assessed the effects of aldrin, dieldrin and endrin on clams or oysters, and on the survival and growth of their larvae. Survival of oyster (*Crassostrea virginica*) larvae was decreased by 0.025 ppm endrin or dieldrin; eggs developed normally at less than 0.1 ppm endrin and 0.05 ppm dieldrin. For clams (*Mercenaria mercenaria*), larval survival was decreased by 0.25 ppm aldrin, but eggs developed normally in 0.5 ppm.

Phytotoxicity--In plants, high levels of aldrin were reported to inhibit the growth of beans and tomatoes while stimulating cucumbers, carrots, and parsnips, but the effects were limited to potted plants and virtually disappeared in the field (Dennis and Edwards 1969). At agricultural levels, aldrin, dieldrin, toxaphene and chlordane were reported to inhibit the growth of corn, soybeans, beans and cotton potted in clay loam (Diaz-Mena 1954). Shaw and Robinson (1960) found tomatoes to be uninhibited by 10 to 100 lbs/A aldrin (11.2-112 kg/ha), but inhibition occurred at 150 lbs/A (168 kg/ha) of aldrin or 200 lbs/A (224 kg/ha) dieldrin. *Vicia faba* was reportedly not inhibited by 25 mg/kg dieldrin in clay soil, but 500 mg/kg caused some stunting (Selim et al. 1970). In general, phytotoxic reactions were more likely to occur in poor than in rich soils, and crop quality often deteriorated before either growth or yield were affected (Roswell 1955).

Fish and amphibians--The 24-hour LC_{50} of aldrin ranged from 0.036 ppm in rainbow trout (*Salmo gairdnerii*) to 0.096 ppm in bluegills (*Lepomis macrochirus*). The96-hour LC_{50} in bluegills was only 0.013 ppm. The 24-hour LC_{50} of dieldrin for rainbow trout was 0.05 ppm, for bluegills 0.0055 ppm; for endrin, the levels were 0.0018 ppm in rainbow trout and 0.0003 ppm in bluegills. The 96-hour LC_{50} of dieldrin in bluegills was 0.008 ppm; of endrin, 0.0006 ppm (Pimentel 1971). The margin between safety and lethality in juvenile spot fish (*Leiostomus xanthurus*) was less than 0.1 ppb: fish exposed to 0.05 ppb were not noticeably affected after three weeks while fish exposed to 0.15 ppb died (Pimentel 1971). Endrin contamination of river basins was strongly correlated with major fish kills between 1963 and 1964, and the fish kills stopped when the endrin levels fell (Breidenbach et al. 1967).

Application of 0.1 to 0.5 lb/A (0.11-0.56 kg/ha) aldrin, endrin, dieldrin, or heptachlor exterminated frogs and toads (Mulla 1962). Tolerance in anurans from heavily treated cotton fields has been reported (Ferguson and Gilbert 1967) and freshwater invertebrates were also observed to develop tolerance (Naqvi and Ferguson 1968). Bioaccumulation in fish has been reported repeatedly (Reinert 1970, Morris and Johnson 1971, Ettinger and Mount 1967) and a twenty-thousand-fold concentration over aqueous levels was induced in shiners under laboratory conditions (Reed 1969).

Birds--In birds, toxicity is often expressed by reproductive failure; these data are reviewed by Stickel (1973), Edwards (1973), and Menzie (1972). Direct toxicity in wild birds has been reviewed by Tucker and Crabtree (1970), Stickel et al. (1969), and Heath et al. (1972), among others. Acute toxicity in chickens ranged from an LD_{50} of 2.7 mg/kg for isodrin to 43 mg/kg for dieldrin (Sherman and Rosenberg 1953) with a 90 percent mortality after 42 days at 12 ppm dietary endrin or isodrin (Sherman and Rosenberg 1954).

Mammals--The acute toxicity of the cyclodiene insecticides to mammals is sufficiently great to warrant their use in vole extermination programs (Schindler et al. 1966) and field mouse control (Lang and Cruger 1960). Their relative toxicity in mammals is: endrin, isodrin > aldrin, dieldrin.

The acute oral LD_{50} of aldrin and dieldrin was between 20 and 70 mg/kg in twelve species (Hodge et al. 1967); isodrin and endrin had an LD_{50} of three to 20 mg/kg in rats (Jones et al. 1968, Servintuna 1964) and were more toxic dermally and by inhalation than aldrin or dieldrin (Spynu 1964). The acute dermal toxicities in rats were estimated at 35 mg/kg for isodrin, 98 mg/kg for aldrin, and 90 mg/kg for dieldrin (Servintuna 1963). Dermal toxicity of heptachlor was 195 mg/kg, and of chlordane 840 mg/kg, in the same study. The acute dermal toxicity of endrin was given as 60-120 mg/kg (Jones et al. 1968). Age affected canine sensitivity to aldrin and dieldrin (Cleveland 1966), and sex affected metabolism and excretion of dieldrin in rats (Klevay 1970). In humans occupationally exposed to aldrin, dieldrin, and endrin, blood levels of up to 0.2 ug/ml for dieldrin and 0.05 to 0.1 ug/ml endrin were said to have no persistent adverse effects on health; symptoms of intoxication, if not fatal, were said to be reversible in a maximum of weeks (Jager 1970, 1971). The general applicability of these data is suspect, however, since all workers with symptoms of organochlorine poisoning were promptly removed from exposure, and all workers with conditions which might predispose to irreversible effects were removed from the areas of exposure. Finally, any workers who were even concerned about possible consequences were removed from the set of exposed workers (Jager 1970, 1971). Thus, the final sample consisted of those workers who were selected for minimum susceptibility to organochlorine poisoning.

The toxicity of cyclodiene insecticides was altered by the presence of other chlorinated hydrocarbon pesticides (Keplinger and Deichmann 1967), organophosphate insecticides (Triolo and Coon 1966) and carbamate insecticides (Williams *et al.* 1967). DDT and dieldrin interacted to increase DDT storage in rats (Street and Chadwick 1967) but decreased DDT storage in guinea pigs (Wagstaff and Street 1971). Aldrin was reported to synergize with other agents in the induction of cardiovascular pathologies (Kagan *et al.* 1974). Dieldrin at ten ppm also decreased the levels of vitamin A in both the maternal and fetal livers of rats (Phillips and Hatina 1972). One ppm of dieldrin reportedly caused an increase in liver weight in female rats after two years (Walker *et al.* 1969), a level which assumes some significance when it is recalled that the ordinary human carries 9.7 to 27 ppm dieldrin in his body fat (Deichmann and MacDonald 1971). Other effects of low levels of dieldrin in experimental animals included irritability in rats fed ten ppm for eight weeks (Walker *et al.* 1969). In a survey of terminal hospital patients, dieldrin levels were significantly elevated in patients with hypertension (Radomski *et al.* 1968).

Aldrin, dieldrin, and endrin were teratogenic in mice and in hamsters; in hamsters, a considerable embryonic mortality was also seen (Ottolenghi *et al.* 1974). At considerably lower levels, Chernoff *et al.* (1975) did not observe and gross malformations in mice, but noted that ossification was retarded and the frequency of extra ribs increased. Endrin was found to be teratogenic in rats and in mice (Noda *et al.* 1972), and endrin and dieldrin adversely affect murine reproduction (Good and Ware 1969). Dieldrin at 0.5 to 2.0 lbs/A (0.56-2.2 kg/ha) had no significant effects on reproduction in penned cottontails (Malecki *et al.* 1974), but was transported into both blastocysts and implanted fetuses in rabbits (Hathaway *et al.* 1967). Aldrin at 0.15 to 0.3 mg/day for fourteen months adversely affected estrus, pregnancy, livebirths, and milk production in females, as well as libido in male beagles, although neither parental illness nor teratogenesis were observed (Deichmann *et al.* 1971).

Aldrin and dieldrin increased the incidence of liver tumors in mice (Davis and Fitzhugh 1962), rats, and dogs (Fitzhugh *et al.* 1964). The significance of these data were initially disputed (Terracini 1967, Barnes 1966, Deichmann _et al_. 1970). More recent data, however, have confirmed the carcinogenicity of dieldrin (Walker _et al_. 1969, 1973; Thorpe and Walker 1973, Vettorazzi 1975).

Mutagenic effects have been suggested for the cyclodiene insecticides in general (Rossival 1970), and chromosome abberations have been induced by endrin in barley (Wuu and Grant 1966). Dean, Doak, and Somerville (1975) reported, however, that HEOD, the pure form of dieldrin was not mutagenic in the host-mediated assay in mice, the dominant lethal test in mice, or in chromosome analyses of hamster cells.

Chlordecone and Mirex

Chlordecone is the common name for 1,1a,3,3a,4,5,5,5a,5b,6-decachloro-
octahydro-1,3,4-methano-2H-cyclobuta(cd)pentalen-2-one, introduced by
Allied Chemical Corp. in 1958 as an insecticide and possible fungicide.
It is synthesized by the condensation of two molecules of hexachloro-
cyclopentadiene in the presence of sulfur trioxide to form mirex, fol-
lowed by hydrolysis to the ketone, chlordecone. It is a stable tan to
white solid of the cyclodiene group of insecticides which sublimes with
some decomposition around 350°C. Its water solubility is 0.4 percent
(4,000 ppm) at 100°C, but it is readily soluble in strongly alkaline so-
lu[illegible] at room temperature and readily forms hydrates at ordinary tem-
peratures and humidity. The technical product is of greater than 90
percent purity.

Mirex, the common name for 1,1a,2,2,3,3a,4,5,5,5a,5b,6-dodecachloroocta-
hydro-1,3,4-metheno-1H-cyclobuta(c,d)pentalene, is a white crystalline
solid with a melting point of 485°C which is insoluble in water and un-
affected by nitric, sulfuric or hydrochloric acids. Introduced by
Allied Chemical Corp. in 1958, mirex is a stomach poison with little
contact toxicity, used primarily in baits against fire ants. It is
produced by the dimerization of hexachlorocyclopentadiene in the pres-
ence of aluminum chloride, but can also be made by reacting chlordecone
with phosphorus pentachloride.

Degradation:

Biological--The single report of the degradation of mirex by sewage
sludge organisms identified neither the organisms nor the metabolites.
The degradation proceeded under anaerobic conditions and was not light-
mediated (Andrade and Wheeler 1974). In estuarine environments, mirex
was converted to chlordecone and reduced chlordecone (Brown et al. 1975).
Essentially no metabolism of mirex was observed in plants. The stability

of ^{14}C-mirex was so great that its recycling after the death of plants was postulated (Mehendale et al. 1972). In rats, mirex was poorly absorbed from the gut, and over 50 percent of ^{14}C-mirex was excreted fecally within 38 hours. Only 0.69 percent of the radioactivity was eliminated as urinary metabolites in the first week, and of the mirex absorbed from the gut, half remained in the tissues after 100 days (Mehendale et al. 1972). In another study, an oral dose of 0.2 mg/kg mirex was excreted unchanged in the feces or stored in the fat in rats. Fecal excretion was 85 percent complete after 48 hours, but after seven days fat levels of mirex were 0.9 ppm, and this level remained constant for at least three weeks. A mirex-photoproduct was equally stable (Gibson et al. 1972).

Chlordecone has not been shown to be degraded by plants, animals, or microorganisms. It is, however, a metabolite of the insecticide Kelevan (decachlorooctahydro-2-hydroxy-1,3,4-metheno-2H-cyclobuta-(cd)-pentalen-2-levulinic acid ethyl ester) in soil and in potato weeds (Sandrock et al. 1974).

Photolysis--Knoevenagel and Himmelreich (1973) reported that photolysis of chlordecone or Kelevan in the presence of oxygen resulted in the formation of carbon dioxide and hydrogen chloride. Data on the conditions or rates of the reactions were not available. Kelevan was converted to mirex by ultraviolet light; it was suggested that the conversion involved splitting of the chlordecone ring to form two half-mirex rings, with subsequent fusion (Begum et al. 1973). Photoproducts of chlordecone hydrate were identical with a mirex photoproduct (Alley and Layton 1974, Alley et al. 1974).

A solution of 0.4 M mirex in 350 ml cyclohexane or isooctane was 95 percent dissipated by ultraviolet light in 48 hours (Alley et al. 1973). On silica gel chromatography plates, mirex in the presence of sun or ultraviolet light decomposed extremely slowly to chlordecone hydrate (Ivie et al. 1974).

Physical or chemical--No data were available on the physical or chemical degradation of mirex or chlordecone, except that mirex is unchanged after treatment with nitric, sulfuric and hydrochloric acids. However, chlordecone appears to decompose somewhat as it sublimes at 350°C (Brooks 1974).

Transport-

In an estuarine environment, 95 percent of the mirex was adsorbed to organic matter, kaolinite, or montmorillonite (Brown et al. 1975). In soil cylinders 80 cm deep, 1.2 percent of the chlordecone leached through clay loam, 17.2 percent leached through clay, 17.4 percent through sandy clay loam, 28.1 percent leached through sandy loam, and 36.8 percent leached through sandy clay loam (Gawaad et al. 1971). Following a single aerial application of mirex as a fire-ant bait, pond sediments contained 0.7 to 1.1 ppb, while soils contained up to 2.5 ppb. Pond water contained 0.2 to 0.51 ppb immediately after treatment, and all residues had disappeared from the water within three months. Bahia grass in the same pond contained mirex residues in roots and blades (Spence and Markin 1974). Mangrove seedlings (*Rhizophora mangle*) contained mirex after the soil was treated with 11.2 kg/ha (Walsh et al. 1974) and apples retained 0.3 ppm chlordecone three months after a spraying resulted in initial residues of 1.6 ppm (Brewerton and Slade 1964).

Four applications of 1.25 lbs/A mirex bait, equalling 1.7 g/A (0.04 kg/ha) of the active ingredient, resulted in residues of 0.65 ppm in catfish after six months, apparently due to uptake during normal feed- ing rather than by direct ingestion of the bait (Collins et al. 1973). In a survey of woodcocks (*Philohela minor*) in southern states, residues of mirex were found in four of ten birds from Mississippi, one of which contained 26.7 ppm. In Louisiana one of ten birds contained mirex as did one of five from Maryland; two of five from Alabama, and two of five from Tennessee (Clark and McLane 1974).

While these data are sparse, it is apparent that mirex and chlordecone, like all the chlorinated hydrocarbon insecticides examined, are transported into all phases of the environment. No data are available for chlordecone in the aquatic-terrestrial model ecosystem, but mirex was among the least degradable compounds tested, with over 97 percent of the extractable radioactivity in the organisms (snail, mosquitos, algae, and fish) being undegraded mirex. Data were also cited on the presence of mirex in fish from Lake Ontario and of up to 20 ppm in wild rodents (Metcalf and Sanborn 1975).

Persistence--

No estimates of the persistence of mirex or chlordecone in soil, and no description of their probable fate in soil, were available. The absolute lack of data on their degradation by microorganisms suggests that mirex and chlordecone may exceed even dieldrin in environmental stability.

Effects on Non-Target Species--

Microorganisms--The effects of chlordecone on microorganisms and soil processes in Egyptian soils were not considered severe. Levels of ten kg/ha did not significantly depress fungi at any time, and even increased their numbers after 45 days; bacterial levels increased for no more than 30 days. Treatment with 22 kg/ha led to inhibition of virtually all soil processes at some time during the two month observation period; actinomycetes were also inhibited (Gawaad *et al.* 1972a, 1972b, 1973a, 1973b). These data are summarized in Table 51, as are the even more sparse data for mirex.

Invertebrates--Use of mirex in baits against imported fire ants did not harm bee colonies near or within treated areas (Glancey *et al.* 1970). The LC_{50} of mirex for red crayfish (*Procambarus clarki*) was greater than 0.1 ppm (Muncy and Oliver 1963). Mortality from water levels of one ppm was delayed, but ingestion of one or two grains of bait caused

Table 51. EFFECTS OF CHLORDECONE AND MIREX ON SOIL PROCESSES AND MICROORGANISMS

Parameter	Effect	Level Original	PPM	# days Observed	Soil or conditions	Reference
Chlordecone						
CO_2 production	+	10 kg/ha	5	7–30	soil, UAR	Gawaad et al. 1972a
	–	22 kg/ha	11	7	" "	Gawaad et al. 1973a
Ammonification	–	10	5	15	" "	Gawaad et al. 1972b
	–	22	11		" "	Gawaad et al. 1973b
Nitrification	–	10	5	15	" "	Gawaad et al. 1972b
	–	22	11		" "	Gawaad et al. 1973b
Bacteria	+	10	5	<30	" "	Gawaad et al. 1972a
	0	10	5	>30	" "	Ibid.
Fungi	0	10	5		" "	Ibid. Gawaad et al. 1973a
Actinomycete	–	10	5	15	" "	Gawaad et al. 1972b
	0	10	5	45	" "	Ibid.
Nitrobacter	–	10	5	60	" "	Gawaad et al. 1972b
Nitrosomonas	0	10	5	60	" "	Ibid.
Mirex						
Saccharomyces cerevisiae	0					Nelson and Williams 1971
Tetrahymena pyriformis	–	.09 µg/l	0.09	7	culture	Cooley et al. 1972
Bacteria	0	1000 ppm	1000		culture	Brown et al. 1975

+ = stimulation 0 = no effect – = inhibition

55.5 percent and 100 percent mortality, respectively, within four days (Ludke *et al.* 1971). If bait was present in the water but inaccessible, crayfish accumulated 1.45 ppm (Ludke *et al. op. cit.*). Nonetheless, Markin *et al.* (1972) concluded that the fire ant program posed no hazards to the Louisiana crayfish industry. No data were available for chlordecone.

Fish and amphibians--Mirex at five ppm, but not at one or three ppm, significantly reduced the growth of bluegills, *Lepomis macrochirus* (Pimentel *et al.* 1971). No mortality and no malformations were observed in caged channel catfish exposed to 1.7 g/A (0.04 kg/ha) of mirex which had been applied as bait at 1.25 lb/A (Collins *et al.* 1973).

Chlordecone inhibited oxygen uptake by bluegill liver mitochondria *in vitro* (Hiltibran 1974) and inhibited ATPase activity in channel catfish (Desaiah and Koch 1975).

Birds--The acute oral LD_{50} of mirex in mallards was greater than 2,400 mg/kg (Tucker and Crabtree 1970). The LC_{50} for pheasants was 1,400 to 1600 ppm; for coturnix quail, over 10,000 ppm, when birds were fed treated feed for five days followed by clean feed for three days (Pimentel 1971). Feeding 440 ppm chlordecone was lethal to adult male quail within seven to 15 days (McFarland and Lacy 1969). The toxicity of mirex to immature quail, but not to adult quail, increased if the photoperiod was shortened (Eroschenko and Wilson 1974). Also in quail, chlordecone induced fatty liver degeneration (Atwal 1973), increased the weight of the adrenal glands, and exerted an estrogenic effect, on both sexes (Eroschenko and Wilson 1974, McFarland and Lacy 1969, Foster 1974). Chlordecone also increased the thickness of eggshells pathologically in wild birds (Erben 1972) and was teratogenic in pheasants and quail (Foster 1974). Mirex at 500 ppm was not estrogenic in quail, pheasants, mallards, or chickens (Foster 1974).

Mammals--The acute oral LD_{50} of mirex to female rats was 365 mg/kg, with 95 percent confidence limits of 281 to 475 mg/kg (Gaines and Kimbrough 1970). The acute oral LD_{50} of chlordecone for rats was 114 to 140 mg/kg (Jones *et al*. 1968); Gaines (1969) cited 125 mg/kg, with 95 percent confidence limits of 115 to 136 mg/kg. Mirex was far more toxic as a five percent solution in corn oil than as a 20 percent corn oil suspension (Gaines 1969).

A chronic intake of five ppm mirex increased parental mortality and decreased litter size in inbred Balb/c mice; in CFW mice, parental mortality was not increased, but litter size decreased (Good *et al*. 1965, Ware and Good 1967). When Balb/c mice were fed ten ppm chlordecone, litter size as well as the number of litters produced decreased and at 40 ppm, females produced no litters. Reproduction resumed seven weeks after chlordecone was removed from the diet, but the first subsequent litters were small, even if treated males were mated with untreated females; if treated females were mated to untreated males, the second litters were also small (Huber 1965). Mirex reduced litter size in rats at 25 ppm but not at five ppm. Mortality of pups born to and nursing on treated dams was higher than if pups nursed on untreated females (Gaines and Kimbrough 1970). Neither mirex nor chlordecone has been proved teratogenic in mammals (Nishimura 1973) but mirex caused cataracts in pups born to and nursing on females fed 25 ppm (Gaines and Kimbrough 1970).

Chlordane

Chlordane is the common name for 1,2,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindene, introduced as a non-systemic contact insecticide by the Velsicol Corporation in 1945 as Octachlor. Two isomers of chlordane have been isolated: *alpha*-chlordane, the *endo-cis* isomer, and *beta*-chlordane, the *endo-trans* isomer. The *alpha*-isomer is more readily dehydrochlorinated and is the chief constituent of the commercial product called *gamma*-chlordane. The melting point of both the *alpha* and the *beta* isomers is between 103 and 105°C.

Chlordane is manufactured by chlorinating cyclopentadiene to give hexachlorocyclopentadiene and condensing the latter with cyclopentadiene to produce chlordene. Chlordene is further chlorinated to chlordane. Chlordane is formulated as 50 and 70 percent emulsifiable concentrates, two and 20 percent kerosene solutions, or five and ten percent dusts and granules. A high purity chlordane, consisting of 95 percent *alpha* and *beta* chlordane, is marketed under the name HCS 3260. It has a water solubility of 56 ppb.

Technical chlordane is a viscous amber liquid (75 to 120 centistokes at 130°F) which is almost insoluble in water but soluble in most organic solvents including petroleum oils. The refined product has a vapor pressure of 1×10^{-5} mm mercury at 25°C. Technical chlordane consists of 60 to 75 percent isomers of chlordane and 25 to 40 percent of related compounds, including two heptachlor isomers.

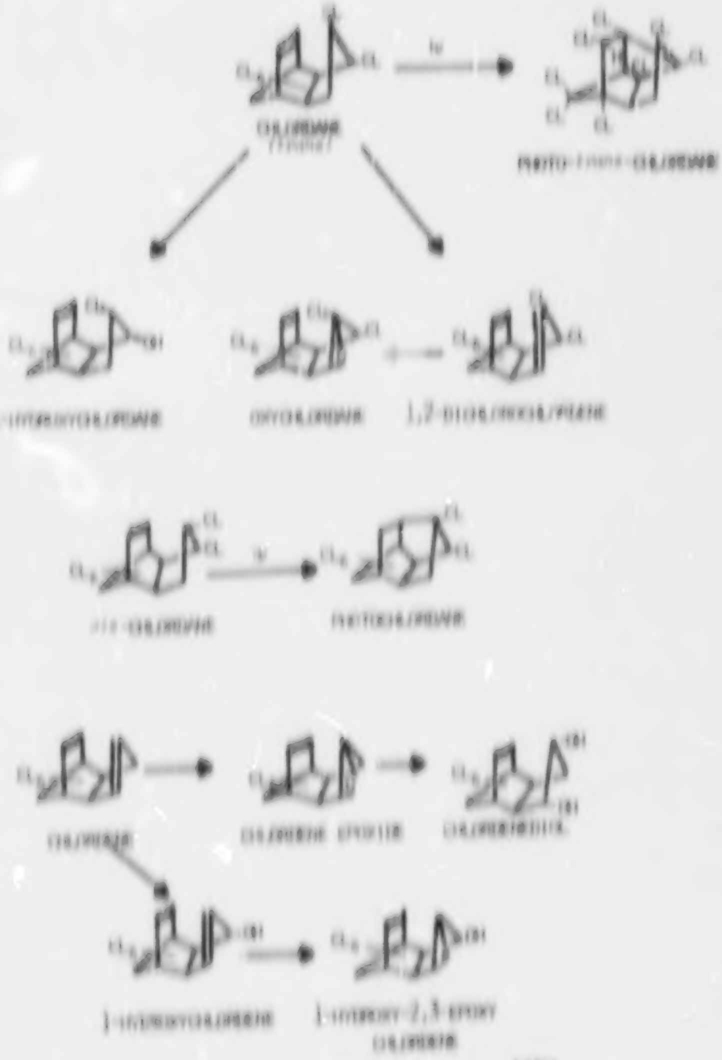
Heptachlor is the common name for 1,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene. It was initially isolated from technical chlordane and was introduced as an insecticide by Velsicol in 1948 as Heptagran, a non-systemic stomach and contact insecticide with some fumigant action. It is a white crystalline solid with a mild camphor odor, which has a water solubility of 56 ppb and a vapor pressure of

3 x 10^{-4}mm mercury at 25°C, and a melting point of 95° to 96°C. The technical product, consisting of approximately 72 percent heptachlor and 28 percent related products, is a soft waxy solid which melts between 46 and 74°C and has a viscosity of 50 to 75 centipoises at 90°C. It is stable to light, air, moisture, and moderate heat, but susceptible to epoxidation. Heptachlor is formulated as an emulsifiable concentrate, a wettable powder, a dust, and a granule. It is synthesized by the action of sulfuryl chloride in chlordene in the presence of benzoyl peroxide, or by the chlorination of chlordene in the dark in the presence of fullers' earth (Martin 1968).

Degradation-

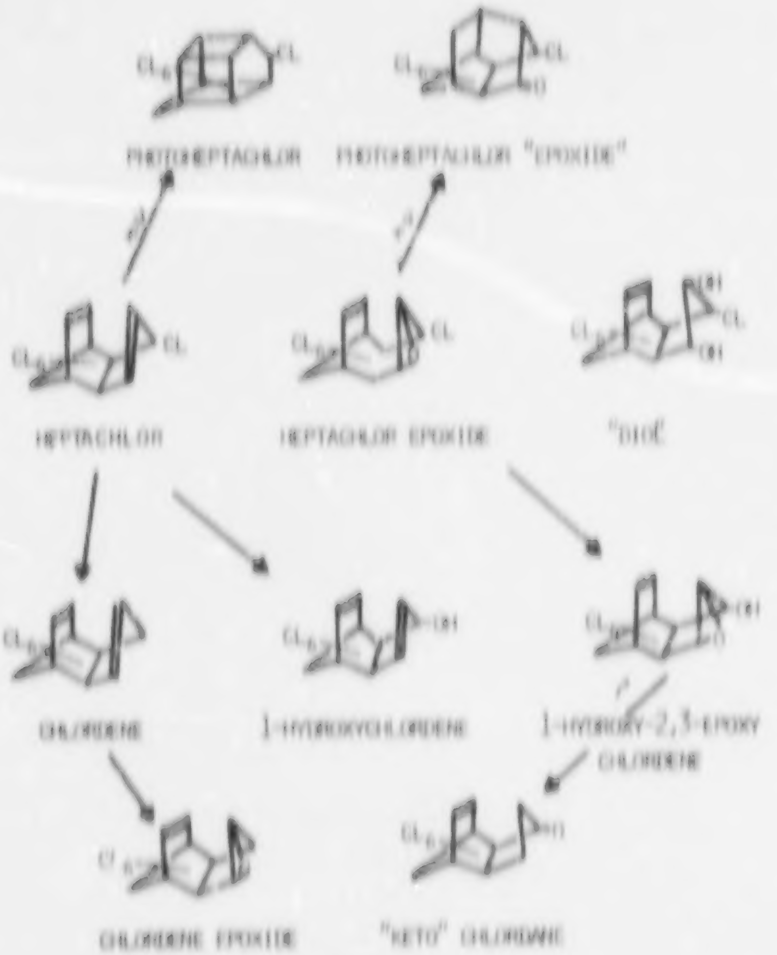
Biological--The few data on microbial degradation of heptachlor and chlordane are shown in Table 52. The degradation pathways of chlordane and heptachlor are shown in Figures 11 and 12, respectively. In one study (Bourquin et al. 1972) $^{14}CO_2$ was recovered from ring-labeled heptachlor and labeling in the cells suggested that additional degradation to CO_2 was masked by incorporation into cellular products. Bourquin and co-workers considered the C-8 of the cyclohexane moiety to be the site of ring cleavage. The most likely compound to be so cleaved was 1-hydroxychlordene, but other substrates were not ruled out. Chlordane, heptachlor epoxide, and 1-hydroxy-2,3-epoxychlordene were also recovered. Castro and Yoshida (1971) reported the total degradation of heptachlor, without formation of heptachlor epoxide, in two months in flooded soils in the laboratory, but no products were identified. Chlordane was metabolized little, if at all, in either flooded or upland soils (Castro and Yoshida 1971). Heptachlor was reported to degrade slowly but steadily in a sewage lagoon but no metabolite characterization was reported (Halvorson et al. 1971).

Heptachlor was epoxidized by 35 of 47 species of fungi and by 26 of 45 species of bacteria and actinomycetes. Heptachlor was metabolized to chlordene, chlordene epoxide and 1-hydroxy-2,3-epoxychlordene via 1-hydroxychlordene (Miles et al. 1969). In sandy loam, the conversion of


CHLORDANE
(trans)
hv
PHOTO-trans-CHLORDANE
1-HYDROXYCHLORDANE
OXYCHLORDANE
1,2-DICHLOROCHLORDENE
cis-CHLORDANE
hv
PHOTOCHLORDANE
CHLORDENE
CHLORDENE EPOXIDE
CHLORDENEDIOL
1-HYDROXYCHLORDENE
1-HYDROXY-2,3-EPOXY
CHLORDENE
Cl
OH

PRODUCTS OF CHLORDANE AND CHLORDENE (AFTER MENZIE, 1974)

Figure 11


CL6
CL
O
PHOTOHEPTACHLOR
PHOTOHEPTACHLOR "EPOXIDE"
hν
hν
HEPTACHLOR
HEPTACHLOR EPOXIDE
OH
"DIOL"
CHLORDENE
1-HYDROXYCHLORDENE
1-HYDROXY-2,3-EPOXY CHLORDENE
hν
CHLORDENE EPOXIDE
"KETO" CHLORDANE

PRODUCTS OF HEPTACHLOR
(AFTER MENZIE, 1974)

Figure 17

Table 52. DEGRADATION OF HEPTACHLOR AND CHLORDANE BY MICROORGANISMS UNDER LABORATORY CONDITIONS

Organism or conditions	Product	Reference
Heptachlor:		
Pseudomonas and mixed cultures	1-hydroxychlordene and some CO_2	Bourquin *et al.* 1972
Clostridium	not identified	Sethunathan and Yoshida 1973
Soil bacteria	chlordene, chlordene epoxide	Miles *et al.* 1969
Fungi, actinomycetes	heptachlor epoxide	Ibid.
Mixed soil microorganisms	1-exohydroxychlordene, chlordene	Miles *et al.* 1971
Sewage lagoon	unidentified	Halvorson *et al.* 1971
Flooded Phillipine soils	not heptachlor epoxide	Castro and Yoshida 1971
Soil	heptachlor epoxide	Gannon and Bigger 1958
Chlorella pyrenoidosa	heptachlor epoxide and 4 minor unidentified products	Elsner *et al.* 1972
Chlordane:		
Aspergillus niger		Iyengar and Rao 1973

heptachlor epoxide to the less toxic 1-*exo*hydroxychlordene was only one percent completed in 12 weeks (Miles et al. 1971).

On cabbage leaves, heptachlor was epoxidized with subsequent formation of other metabolites which included hydroxychlordene and prilic acid. Methoxychlordane and hydroxydihydroheptachlor were found in the soil (Weisgerber et al. 1974). Kaul et al. (1972) identified the major metabolite of *trans*-chlordane on cabbage as 2,4,5,6,7,8,8-heptachloro-2,3,3a,4,7,7a-hexahydro-4,7-methano-1H-inden-1-ol ten weeks after application of chlordane. Peanuts grown on soil treated with heptachlor contained heptachlor epoxide (Morgan et al. 1967). The green alga, *Chlorella pyrenoidosa*, metabolized heptachlor to heptachlor epoxide and 4,5,6,7,8,8-hexachloro-2,3-epoxy-4,7-methano-3a,4,7,7a-tetrahydroindan-1-one with heptachlor epoxide representing 68 percent of the algal associated radioactivity (Elsner et al. 1972).

Heptachlor is metabolized to heptachlor epoxide in rats, dogs, rabbits (Brooks 1969), and cows (Davidow et al. 1953, Gannon and Decker 1960). Mizyukova and Kurchatov (1970) injected rats intraperitoneally with 120 mg/kg of heptachlor, and noted that heptachlor was present in liver, fat, and blood in one hour and heptachlor epoxide was present in the same tissues within four hours. When 25 ug of ^{14}C-heptachlor were injected intravenously, most of the radioactivity was excreted in the feces, partly as heptachlor epoxide and partly as an unidentified urinary compound which, in rabbits, was tentatively identified as hydroxychlordene epoxide (Brooks 1969). Female rats epoxidized heptachlor more slowly than did males, and were able to accumulate more heptachlor epoxide with less toxicity than were males (Radomski and Davidow 1953). When *beta*-hydroxyheptachlor was injected intravenously into rats, 36 percent of the radioactivity was excreted within 48 hours (Korte et al. 1970).

Barnett and Dorough (1974) analyzed chlordane metabolism in rats. When a single oral dose of radioactive chlordane was given, more than 90 percent was eliminated within one week. *Cis*-chlordane was eliminated somewhat more quickly than *trans*-chlordane (70 versus 60 percent in 24 hours). Of the total radioactivity eliminated in the first 24 hours, 13 percent was eliminated as unmetabolized chlordane, primarily in the feces. If chronic levels of one, five, or 25 ppm were fed, fat storage after 56 days was equal to three times the dietary intake, and oxychlordane was the major and most persistent tissue residue. Oxychlordane was eliminated in the feces if fed directly, but was not found as a fecal metabolite of either *cis*- or *trans*-chlordane. The excretion of chlordane decreased when feeding ended.

Photolytic—

Numerous photoproducts of heptachlor and chlordane have been identified (Ivie et al. 1972, Knox et al. 1973, Parlar and Korte 1973, Onuska and Comba 1975, Gaeb et al. 1975). Oxychlordane, an oxidative product of chlordane, was shown to isomerize in the presence of xanthene photosensitizers to form a keto-product of considerable murine toxicity, and an epoxy-intact isomer of low toxicity (Ivie 1973). At wave lengths of 254 nm deposits of solid cyclodienes formed bridged photoisomers (Fischler and Korte 1969) while *trans*-chlordane and nonachlor, both constituents of technical chlordane, formed bridged isomers mostly at wavelengths greater than 300 nm (Vollner et al. 1971). Some dechlorination also occurred for chlordane at wavelengths of more than 300 nm. In direct sunlight, chlordane dissipated faster than either DDT or dieldrin with decomposition presumed but not established (Ginsburg 1953). Decomposition of volatilized cyclodienes is given support both by the numbers of photoproducts found (see above) and by the data of Stanly et al. (1971), who found neither heptachlor epoxide nor chlordane in any of 880 composite atmospheric samples.

Chemical and physical—Vollner and Korte (1974) reported 70 percent decomposition of *cis*-chlordane by 54 Mrad of ^{60}Co *gamma*-irradiation when five to ten mg/ml of chlordane were dissolved in hexane, and 0.29 Mrad were delivered per hour. When dissolved in hexane at 0.2 ppm, heptachlor was degraded by 0.5 to 2.5 Mrads of ^{60}Co *gamma*-radiation or by 20 Mrads of *beta*-radiation, but no metabolites were identified (Caurvels et al. 1974).

Chemically, heptachlor dissolved in butanol was degraded to hydroxychlordene by 16 Kbar pressure at 110°C in the presence of sodium hydroxide. The reaction was 60 percent complete in six hours (Roemer-Maehler et al. 1973). Kaneda and co-workers (1974) reported degradation of heptachlor by aqueous saturated solutions of calcium hypochlorite. The only meta- bolite identified was 1-hydroxychlordene, but further degradation occurred. Heptachlor was 80 percent destroyed by five hours exposure to $KMnO_4$, but not by exposure to chlorine gas or alkali (Leigh 1969).

When river water was contaminated with heptachlor, heptachlor epoxide, *alpha* chlordane and *gamma* chlordane, only heptachlor epoxide remained unaffected after eight weeks. Eighty-five percent of the chlordane remained, and no conversion products were identified. Heptachlor was converted to 1-hydroxychlordene and heptachlor epoxide, with an equilibrium ratio of 2:3 achieved in four weeks (Eichelberger and Lichtenberg 1971). The authors considered chemical rather than biological degradation to have effected the changes, but the water was not sterile so that it is impossible to rule out biological degradation. During storage, three percent of a heptachlor dust formulation decomposed in 42 months with no product characterization reported (Raman and Krishnamoorthy 1973).

Transport:

Within soil--Heptachlor applied to soil at one, two or three lbs/A (1.12, 2.24 or 3.36 kg/ha) was found mostly (90 percent) in the top three inches of soil after one year and after three years over 70 percent of the recovered residues were still found in the top three inches of soil (Lichtenstein et al. 1962b). When heptachlor was applied at five lbs/A (5.60 kg/ha) for five years, or once at 25 lbs/A (28.0 kg/ha), residues decreased by 76 to 82 percent if the soil was cultivated. Of the residues recovered, 26 percent were in the top two in., 52 percent were between two and four inches, and 6.5 percent were in the six to nine inch layer of soil (Lichtenstein et al. 1971b). Residues of heptachlor in a sloping field decreased most at high points, and buildup at low points followed plow line, suggesting washoff rather than leaching as the mechanism of transport (Peach et al. 1973). High levels of organic matter in the soil decreased degradation, leaching, and volatilization of heptachlor (Bowman et al. 1965). When applied as a termiticide, heptachlor exhibited little lateral or vertical movement after 15 years (Stewart and Chisholm 1971) or 21 years (Bennett et al. 1974). After four months during the growing season, heptachlor which had been disked 7.5 cm into the soil was found to have migrated to a depth of 30 cm, although 68 percent of the residue had remained *in situ* (Caro 1971). Chekol and Yurovskaya (1967) concluded that heptachlor had a mobility of 21 cm per growing season and was therefore a potential contaminant of water supplies. Granular heptachlor was more mobile in soil than diazinon, parathion or phorate but mobility depended on the type of soil and level of insecticide used, since bioassays were used (Burkhardt and Fairchild 1967).

Between soil and water--In a newly developed Great Plains irrigation district, when heptachlor was applied in such a way that soil residues of the epoxide 1.5 to 2.5 months later were 0.16 to 1.3 ppm, reservoir water was found to contain one ppt of heptachlor and 0.006 ppb of its epoxide (Knutson et al. 1971b). In West Virginia, 35 ponds examined were all contaminated with heptachlor. Water levels ranged from less than one ppb to 291 ppb and levels in mud ranged from less than one ppb to 59.9 ppb which were in all cases lower than soil levels in the surrounding watershed. Detectible residues persisted for 25 months. The uneven distribution of residues in the different ponds was found to be correlated with distance from treated areas rather than with types of soil (Weatherholtz et al. 1967). Among the mechanisms for removal of heptachlor and chlordane from water into sediment are bacterial flocs

(Speidel et al. 1972) and adsorption by suspended particles (Weil et al. 1972).

Volatilization--Both heptachlor and chlordane have a higher vapor pressure than DDT and are correspondingly more volatile on inert surfaces. In the absence of wind, rain, or direct sunlight, heptachlor toxicity was reduced by 41.9 percent in 24 hours (Mistric and Gaines 1953). Over a treated field, 3.9 percent of the applied heptachlor accumulated on fiberglass filters (Caro et al. 1971). Conversely, dissipation of heptachlor and chlordane from alfalfa was 95 percent complete in three weeks, with 60 percent (0.6 ppm from two lbs/A) present in the soil (Dorough et al. 1972).

Bidleman and Olney (1974) concluded that most chlorinated hydrocarbons are airborne as vapor rather than adsorbed to particles. They recorded chlordane levels of 0.25 ng/m^3 over Providence, Rhode Island. Over Oahu (Hawaii), rainwater contained 1-3 ppt chlordane (Bevenue et al. 1972). The purified chlordane, Velsicol HCS-3260, was less volatile than technical chlordane and was highly persistent in soil (Harris 1973).

Into organisms--It has long been known that cows fed heptachlor excreted heptachlor epoxide in their milk (Davidow et al. 1953, Bruce et al. 1966, Dorough and Hemken 1973). Hogs foraging on corn stover in fields previously treated with heptachlor accumulated heptachlor epoxide in their fat (Dobson et al. 1972) and chickens fed 10-15 ppm of chlordane for five days accumulated 10-13.3 ppm in their fat (McCaskey et al. 1968). Indirect contamination of mammals has been observed in the arctic, where polar bears, seals, porpoises, and foxes all contained measurable residues of heptachlor epoxide. Arctic sheep were free from residues of heptachlor epoxide (Clausen et al. 1974).

Hannon et al. (1970) observed a higher ratio of heptachlor to heptachlor epoxide at higher trophic levels in Lake Poinsett, South Dakota; Gish (1970) found *gamma*-chlordane residues in most heptachlor-treated soils. Both studies noted increasing levels of organochlorine contamination (including DDT and several cyclodienes) at higher trophic levels. Gish considered the organochlorine content of some earthworms (13.8 ppm) to be high enough to cause acute poisoning in birds. In Louisiana, spraying of heptachlor between 1956 and 1962 against fire ants resulted in woodcocks contaminated with 2.4 ppm heptachlor epoxide in 1962, but only 0.42 ppm in 1965 (McLane et al. 1971).

The uptake of heptachlor by plants has been reported frequently (King et al. 1966, Waldron et al. 1968, Lichtenstein and Schulz 1965, Bruce and Decker 1966). In potato fields, soil applications of 1.25 to 2.5 kg/ha of heptachlor resulted in tuber residues which exceeded the permissible levels for three years; the structurally similar insecticides aldrin and dieldrin were also taken up (Poltzu et al. 1972). Applications of one or two kg/ha heptachlor at planting resulted in residues of 0.04 mg/kg in potato skin and 0.01 mg/kg in their pulp (Sazonov and Chikhacheva 1972).

Popov and Donev (1970) reported heptachlor uptake into wheat, beans, sugar beets, and oat vetch; Camoni et al. (1971) found residues of 0.03 ppm heptachlor and 0.005 ppm heptachlor epoxide in beet roots when soil levels were 0.017 ppm and 0.024 ppm, respectively (Van Steyvoort 1968). In soybeans, heptachlor residues were concentrated somewhat over soil levels (Beall and Nash 1969). Soybeans planted 15 years after the application of heptachlor still contained detectible residues of heptachlor epoxide (Nash and Harris 1973). Heptachlor is taken up by corn plants less readily than either aldrin or dieldrin, but is translocated more readily within the plant (Poltzu et al. 1971).

The heptachlor residues of corn, oats, soybeans, and peanuts were correlated with their fat content (Bruce et al. 1966). Applications of two to four lbs/A (2.24 to 4.48 kg/ha) of chlordane to corn fields at planting resulted in no detectible residues (< 0.008 ppm) in corn or cobs, and only traces in stalks, at harvest. Silage plants harvested 102 days after planting contained 0.03 to 0.04 ppm of chlordane (Dorough and Pass 1972).

In a sandy Nova Scotia loam soil, chlordane was taken up into parsnips, beets, carrots, potatoes, and rutabagas with carrots containing the highest residues (0.26 ppm). At harvest, 16 months after chlordane treatment, potatoes were found to contain 0.15 ppm, parsnips 0.12 ppm, carrots 0.07 ppm, and rutabagas and beets, 0.01 ppm chlordane residues. The composition of the plant-absorbed residues was similar to the composition of the applied chlordane (Stewart 1975). The high purity chlordane, Velsicol HCS-3260, was taken up by plants in the order: radish > potatoes > carrots > beans. In alfalfa, the residues consisted of *alpha*- and *gamma* chlordane, photo-*alpha*-chlordane, and oxychlordane (Wilson and Oloffs 1976).

A 61-81 percent reduction in crop residues of heptachlor, heptachlor epoxide, and chlordane resulted from the application of carbon at the rate of 2,000 ppm to contaminated soil. The protective effect lasted at least four seasons (Lichtenstein et al. 1968, 1971c). Increasing the interval between treating and planting fields decreased plant uptake of organochlorine insecticides, which included chlordane and heptachlor. Soil differences also affected plant uptake (Douls 1970).

When the fate of heptachlor was investigated in a terrestrial-aquatic model ecosystem, it was found that heptachlor behaved somewhat like aldrin (q.v.), being readily converted to its epoxide. Once formed, heptachlor epoxide was approximately as stable as dieldrin (q.v.). An additional degradative pathway was the conversion of heptachlor to 1-hydroxychlordene, mostly by chemical hydrolysis, resulting in a significant reduction of toxicity and an increase in water solubility. The authors considered heptachlor epoxide to be highly bioaccumulative, but 1-hydroxychlordene and its epoxide were considered not to be truly bioaccumulative. Heptachlor was not converted to chlordene under the conditions of the model ecosystem (Lu et al. 1975).

When the fate of HCS chlordane was investigated in a model terrestrial-aquatic ecosystem, algae accumulated ^{14}C-chlordane 98,386-fold over the concentration found in the water. Magnification by snail, mosquito, and fish was 132,613-fold, 4,132-fold, and 8,258-fold greater than the water levels of ^{14}C-chlordane (Sanborn et al. 1976). While not showing increased uptake of chlordane with increasing trophic level, these data demonstrate the extreme affinity of chlordane for lipid and the insufficiency of water levels of chlordane as a measure of chlordane contamination.

Persistence-

Data on the persistence of chlordane and heptachlor in soils under various conditions are summarized in Tables 53 and 54. In Table 54, heptachlor residues include or are identical with heptachlor epoxide residues.

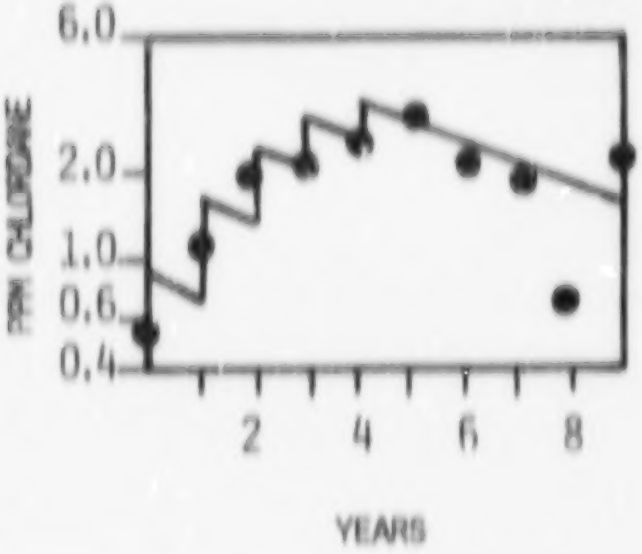
Wingo (1966) argued that accumulation of high concentrations of heptachlor in soil was unlikely at normal agricultural levels, but his data showed that persistence increased with level of application. Figure 13 (after Hermanson et al. 1971), shows the effect of repeated applications of chlordane to soil. Baluja-Marcos and Serrano-Gonzalves (1970) estimated the time required for elimination of both heptachlor and chlordane from Spanish agricultural soils to be between three and five years which compares well with the data of Hermanson et al. (1971) who estimated the so-called persistence-half-life for both compounds to be four years. No change was observed in the levels of heptachlor or untreated soil, autoclaved soil, or either of these ammended with five ppm of urea, in 11 months following application of heptachlor (Kiigemagi et al. 1958). Chlordane was not noticeably degraded under aerobic or anaerobic conditions in various Phillipine soils (Castro and Yoshida 1971). Chlordane and heptachlor retained their termiticidal capacity for up to 21 years, but residue levels were not given (Johnston et al. 1971). When heptachlor was applied at ten lbs/A/yr (11.2 kg/ha) for three years the

Table 53. RESIDUES AND RATES OF DEGRADATION OF CHLORDANE IN SOIL UNDER FIELD CONDITIONS

% residue	# of years	% mean loss/yr	Conditions	Reference
15	21	4.05	applied as termiticide	Bennett *et al.* 1974
9	16	5.69	Congaree sandy loam	Nash and Harris 1973
15	11	5.57	sandy loam	Chisholm and MacPhee 1972
16	15	5.60	sandy loam; incorporated	Stewart and Chisholm 1971
40	14	4.29	maximum persistence	Nash and Woolson 1967
15	12	7.08	turf soil	Lichtenstein and Polivka 1959
2.3	7	13.96	coral; incorporated	Bess and Hylin 1970
2.9	7	13.87	sandy loam; incorporated	Ibid.
2.4	7	13.94	clay; incorporated	Ibid.
50	4	12.50	soil incorporated	Gunther *et al.* 1971
13.8	4	21.55	lawn treatment	Lidgate 1967
15	3	28.33	soil incorporated	Czaplicki 1969

Table 5A. RESIDUES AND RATES OF DEGRADATION OF HEPTACHLOR UNDER FIELD CONDITIONS
RESIDUES INCLUDE HEPTACHLOR EPOXIDE

% residue	# of years	% mean loss/yr	Conditions	Reference
10	16	5.63	Congaree sandy loam	Nash and Harris 1973
16	14	5.25	maximum persistence	Nash and Woolson 1967
12	10	8.80	turf, no cropping; hay cut	Stewart and Fox 1971
4-5	9	10.61	turf	Lichtenstein and Polivka 1959
0	9	≥11.11	turf	Ibid.
17.8	9	9.13	silt loam; dust formulation	Wilkinson *et al.* 1964
31.7	9	7.59	silt loam; dust formulation	Ibid.
0.96	7	14.15	coral, incorporated	Bess and Hylin 1970
1.37	7	14.09	sandy loam, incorporated	Ibid.
2.67	7	13.90	clay, incorporated	Ibid.
4-5	6-10	11.94	loam, 25 lbs/A/year	Lichtenstein *et al.* 1970
50	4	12.50	Beltsville sandy loam	Hermanson *et al.* 1971
10	3	30.00		Czaplicki 1969
[illegible]	3	30.33	soil incorporated	Turner *et al.* 1972
18-25	1	75.5	sandy soil; granule formulation	Sazonov and Chikhacheva 1972
50	0.9	55.55	soil incorporated	Freeman *et al.* 1975



VARIATIONS IN THE TOTAL ORGANICALLY BOUND CHLORIDE CONTENT OF SOIL SAMPLES, EXPRESSED AS PERSISTENCE CURVES

(HERMANSON ET AL. 1971)

Figure 13

residues decreased to two ppm heptachlor, 0.8 ppm heptachlor epoxide, and 2.1 ppm *gamma*-chlordane six years after the first application from a total application of 15 ppm (Stewart and Chisholm 1965).

The insect toxicity of chlordane applied to soil at ten to 15 lbs/A (11.2 to 16.8 kg/ha) was undiminished after three years (Wolcott 1954). Harris (1964a, 1964b, 1967, 1972b) and Harris and Sans (1972) found that the toxicity of chlordane and heptachlor was affected by soil type and soil moisture. The biological activity of heptachlor was in some cases correlated more closely with the organic content of the soil than with the concentrations of pesticide. Field-moist soil (12.1 percent moisture) increased the insecticidal activity of heptachlor more than 12-fold over oven-dried soil (1964b). Heptachlor epoxide was more toxic in moist soil due to fumigant action, and more persistent in mineral soil than heptachlor itself (Harris and Sans 1972). Carbon amendment of soil prevented the extraction of heptachlor from sandy or loamy soil and decreased its toxicity. Binding of heptachlor to carbon increased with time, and was more striking under laboratory conditions than in the field (Lichtenstein et al. 1968). Heptachlor also adsorbed to clays, both on surfaces and into the intralamellar spaces of montmorillonite (Ju-Chang and Liao 1970). Sorption onto kaolinite or montmorillonite in water was slow and required one month to reach equilibrium (Weil et al. 1972).

In discussing the persistence of chlordane and heptachlor, it is necessary to include their conversion products. Few data are available on the products of chlordane in soils, but the longer persistence and high insecticidal activity of heptachlor epoxide has been known for years (Gannon and Bigger 1958). In Queensland soils, the conversion of heptachlor to its epoxide is essentially completed within 43 months (Stickley 1972). Chopra (1966) found that heptachlor epoxide was not formed in sandy loam, but was formed in silt loam. Carter and Stringer (1970) found that up to 60 percent of the heptachlor residues in an Oregon loamy fine sand consisted of 1-hydroxychlordene, and heptachlor epoxide formed only a small part of the residues in most soils. Further analysis of the heptachlor residues in Oregon soils identified the major residues of technical heptachlor as: heptachlor, 1-hydroxychlordene, *gamma*-chlordane and *alpha*-chlordane, nonachlor, and heptachlor epoxide. One to two years later, 1-hydroxychlordene had essentially been replaced by its epoxide, 1-hydroxy-2,3-epoxychlordene with soil type affecting the relative levels of the various residues (Carter *et al.* 1971).

Lichtenstein *et al.* (1970) pointed out that applications of 25 lbs/A (28.0 kg/ha) of technical heptachlor included 6.25 to 7.5 lbs of *gamma*-chlordane and 1.0-2.5 lbs of nonachlor. Ten years later, relatively more *gamma*-chlordane than heptachlor/heptachlor epoxide remained in the soil and residues were claimed to be eight percent of the total technical heptachlor, but no reference was made to hydroxychlordene or its epoxide. These data make it apparent that residues are defined in different ways, adding to the variability imposed by soil, temperature and other climatic variables.

The persistence of heptachlor and chlordane can be gauged, if only indirectly, by the observations that in Ontario all tested soil which had histories of heptachlor administration contained *gamma*-chlordane (Harris *et al.* 1966); of 20 fields in northern Saskatchewan, ten contained heptachlor/heptachlor epoxide residues (Saha *et al.* 1968). In a later study, 17 of 41 soils contained chlordane, and 24 of 41 contained heptachlor or its epoxide, even though samples were not selected by their history of pesticide contamination (Saha and Sumner 1971). Yule, Chiba and Morley (1967) observed no decomposition of soil-incorporated heptachlor in 11 months.

Effects on Non-Target Species:

Microorganisms.--Richardson and Miller (1960) observed that chlordane and heptachlor inhibited mycelial growth in culture. In flask culture, chlordane and heptachlor inhibited nitrification (Winely and San Clemente

1970). As shown in Tables 55, 56 and 57, however, neither chlordane nor heptachlor is excessively destructive of the overall fungal and bacterial populations of soils.

Heptachlor, but not chlordane, enhanced ammonification in soil (Peri 1952, Jones 1956, Gordienko 1964). Both heptachlor and chlordane probably enhance nitrification at least temporarily, even though Peri (1952) found no significant effects on the numbers of nitrifying bacteria when chlordane was applied at agricultural levels. Heptachlor had no effect on ammonifying bacteria in alluvial meadow soil (Rankov and Khristova 1971), but did inhibit nitrifying bacteria in pot culture (Gawaad *et al*. 1973). Preplanting applications of 0.6 kg/ha in the row, or 0.5 kg/ha seed treatment with heptachlor, reportedly increased the numbers of azotobacteria and fungi during the growing season (Khalidov *et al*. 1968).

Gordienko (1964) suggested that the observed stimulatory effects of heptachlor on available nitrate were due to death and decomposition of microorganisms. This hypothesis is supported by Shamiyeh and Johnson (1973) who found that 100 mg/liter (100 ppm) of heptachlor inhibited 89 percent of bacterial cultures, 81 percent of actinomycetes, and 50 percent of fungal cultures on agar. Contamination of agar with 25 mg/liter (25 ppm) of heptachlor killed 63 percent of bacteria transferred onto the plates. Bacterial resistance could be selected for at these levels. Shamiyeh and Johnson further determined that, in soil, total numbers of bacteria increased, with the greatest increase occurring six to nine months after the application of 55 kg/ha. Total numbers of fungi showed an initial decrease. Chlordane was toxic to several pathogenic soil fungi in the laboratory, and sufficiently toxic to *Ophiobolus* (Take-all, on wheat) to be of significance under field conditions. *Pythium*, on the other hand, was stimulated by chlordane (Grossman and Steckhan 1960).

Table 33. THE EFFECTS OF CHLORDANE ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	 PPM	# days observed	Conditions	Reference
Ammonification	0	agri. levels				Peeri 1952
	–	0.01%	100	to 1000	sandy loam, pH 7.6–8	Jones 1956
Nitrification	0	50–200 kg/ha	22–40			Tewari and Chakravarty 1970
	+	50–100 lbs/A	25–50			Pathak et al. 1961
	0	10–100 lbs/A	5–50			Shaw and Robinson 1960
	+	0–200 ppm	0–200			Varade and Ballal 1969
	–			4	flasks	Winely and San Clemente 1970
Available P_2O_5	+		0.2			Varade and Ballal 1969
Bacteria, all	+	50–100 lbs/A	25–50			Pathak et al. 1961
Nitrifying bacteria	0	agri. levels				Peeri 1952
Ammonifying bacteria	0	agri. levels				Ibid.
Bacillus megaterium	–	20 ppm	20		Laboratory	Trudgill et al. 1971
B. subtilis	–		20		–	Ibid.
B. cereus	–		20		–	Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 55. Cont'd

Parameter	Effect	Level Original	 ppm	# days observed	Conditions	Reference
Staphylococcus aureus	-		20		Laboratory	Trudgill et al. 1971
Streptomyces [illegible]	-		20		"	Ibid.
S. [illegible]	-		20		"	Ibid.
[illegible] sp. B	-		20		"	Ibid.
Corynebacterium T_1	-		20		"	Ibid.
Sarcina lutea	-		20		"	Ibid.
Achromobacter	0		20		"	Ibid.
E. coli	0		20		"	Ibid.
Klebsiella	0		20		"	Ibid.
Penicillium (7 sp.)	0		20		"	Ibid.
Phytoplankton	-	1 ppm	1	1/6		[illegible] and [illegible] 1970
Fungi	+	50-100 lb/A	25-50			Pathak et al. 1961
[illegible]	-	agri. levels				[illegible] 1952

+ = stimulation
0 = no effect
- = inhibition

Table 56. THE EFFECTS OF HEPTACHLOR ON SOIL PROCESSES

Parameter	Effect	Level original	Level PPM	# days observed	Conditions	Reference
Ammonification	+	12,24 kg/ha	6,12		soil	Gordienko 1964
Nitrification	–	12,24 kg/ha	6,12		soil	Ibid.
	–			4	flasks	Winely and San Clemente 1970
	+	.05%	500	30		Brown 1954
	0	.05%	500	60		Ibid.
	0	30-300 lbs/A	15-150			Shaw and Robinson 1960
Catalase activity	–			80	boggy meadow	Tsirkov 1969
Urease activity	–			80		Ibid.
Protease activity	0			80		Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 57. THE EFFECTS OF HEPTACHLOR ON SOIL MICROORGANISMS

Parameter	Effect	Level original	ppm	observed	Conditions	Reference
Bacteria: all	−			56		Chandra 1967
	−	100 mg/l	100		on agar	Shamiyeh and Johnson 1973
	−	25 mg/l	25		on agar	Ibid.
	+	55.3 kg/ha	~27	270	on soil	Ibid.
	+	3200 mg/kg	3200	77	on soil	Ibid.
Ammonifying	0	40-60 mg/kg	40-60	40	alluvial meadow	Rankov and Khristova 1971
Nitrifying	−	10-50 ppm	10-50		pots	Gawaad et al. 1973
Nitrobacter	0			9	flasks	Winely and San Clemente 1970
Fungi	+	agri. levels			on agar	Shamiyeh and Johnson 1973
	−	100 mg/l	100		on agar	Ibid.
Actinomycetes	−	100 mg/l	100		on agar	Ibid.
	0	40-60 mg/kg	40	40	alluvial meadow	Rankov and Khristova 1971
Aquatic micro-organisms	−	1 ppm	1	1/6	water	Ware and Roan 1970
phytoplankton	−	.001 mg/l	.001		lethal level	Kenaga 1970
zooplankton	−	.01 mg/l	.01		lethal level	Ibid.

+ = stimulation
0 = no effect
− = inhibition

These data, and the species examined for chlordane susceptibility by Trudgill et al. (1971) as listed in Table 55 argue that heptachlor and chlordane are toxic to some but not all microorganisms, so that major changes in the composition of soil microbial populations must be expected in the presence of these compounds, even if overall numbers of microorganisms remain constant.

Plants--Stitt and Evanson (1949) reported that 34.8 lbs/A (38.9 kg/ha) of chlordane adversely affected cucumbers, bush beans, and turnips. Boswell (1955) noted that phytotoxicity was more likely in poor soils than rich, presumably because the latter have greater adsorptive capacity. In quartz sand chosen for its lack of adsorptive capacity, chlordane significantly reduced the growth of corn roots, but not of pea roots while heptachlor increased root growth of both corn and pea roots, the latter significantly (Lichtenstein et al. 1962). Shaw and Robinson (1960) found that 200 lbs/A of chlordane (224 kg/ha) or 120 lbs/A of heptachlor (134.4 kg/ha) were required to inhibit the growth of tomatos or sudan grass.

These data suggest that, except under conditions of extreme soil poverty and gross contamination, neither heptachlor nor chlordane is likely to exert direct toxic effects on plants. Even in the absence of pests, heptachlor reportedly increased yields of several crops (sugar beets, winter rye, spring wheat, barley, oats, potatoes) for two years after a single application. The crop improvement was synergistic with mineral fertilizers (Persin 1961).

Invertebrates--The effects of heptachlor and chlordane on soil fauna were reviewed by Edwards and Thompson (1973) who concluded that chlordane and heptachlor drastically decrease the numbers of earthworms; considerably decrease the numbers of *Collembola*, paurapods and soil mites. They may also kill some terrestrial molluscs, but do not much affect springtails or symphelids. Heptachlor was reported to decrease the numbers of *Acarinae* and increase the numbers of *Collembola* when applied to a beet field at 0.6 kg/ha (Khalidov _et al_. 1968).

Freshwater invertebrates from areas contaminated by chlordane were more resistant to the pesticide than those from uncontaminated areas (Naqvi and Ferguson 1968). Chlordane at 0.01 ppm reduced shell growth and shell movement in oysters (Butler _et al_. 1960).

Fish--The LC_{50} for various fish to chlordane ranged from 0.01 ppm in 48 hours for rainbow trout to 0.082 ppm for goldfish after 96 hours (Pimentel 1971). For heptachlor, the LC_{50} was 0.25 ppm after 24 hours, but 0.009 ppm after 96 hours in rainbow trout while goldfish had a 96 hour LC_{50} of 0.23 ppm (Pimentel 1971).

In mummichogs (*Fundulus heteroclitus*) heptachlor toxicity was greater after 24 hours than after 96 hours, either as a result of bioaccumulation or because of the toxicity of heptachlor epoxide (Eisler 1970a). Nevertheless, heptachlor was among the least toxic organochlorines (Table 36). Khan and co-workers (1973) determined the relative toxicities of chlordane and heptachlor in fish to be less than those of chlordane and photoheptachlor, respectively.

Amphibians--Heptachlor, but not chlordane, was toxic to toads (*Bufo boreas*) and frogs (*Scaphiopus hammondii*) at 0.1 to 0.5 lbs/A (0.11 to 0.56 kg/ha) (Mulla 1962). The 24 hour LC_{50} of heptachlor for Fowler's toad was 0.85 ppm (Pimentel 1971).

Birds--Tucker and Crabtree (1970) listed the acute oral LD_{50} of chlordane and heptachlor to mallards as 1,200 mg/kg and more than 2,000 mg/kg, respectively. Coturnix quail which were fed 0.05 to 0.1 mg heptachlor per day for 18 to 32 days produced normal numbers of eggs of the normal weight. Prehatching mortality among the chicks was also normal, but deaths among newly-hatched chicks were higher for the first week after hatching. Survivors were normal with respect to survival, age of sexual maturity, and fecundity. Egg residues of heptachlor ranged from one to 17 ppm (Grolleau and Froux 1973). When applied to fields at agricultural levels, heptachlor has repeatedly been shown to reduce bird populations as reviewed by Pimentel (1971).

When chlordane in acetone was injected into hens' eggs, no increase in mortality occurred at levels up to 500 ppm. Heptachlor at 400 ppm and 500 ppm killed 20 and 47 percent of the chick embryos, respectively (Dunachie and Fletcher 1969). When 0.1 ml of one percent heptachlor was injected into the yolk sacs of fertile hens' eggs before incubation, the number of eggs which hatched was reduced to 65 percent of control levels (McLaughlin *et al.* 1962).

Mammals--Estimates for the acute oral LD_{50} of chlordane in rats include 283 mg/kg (Jones *et al.* 1968) and 335 mg/kg in males, 430 mg/kg in females (Gaines 1969) and 560 mg/kg (Spynu 1964, Martin 1968). In mice, Spynu (1969) calculated the acute oral LD_{50} to be 220 ± 34 mg/kg. Heptachlor was calculated to have an acute oral LD_{50} of approximately 100 mg/kg in rats (Martin 1968, Gaines 1969); female rats were less sensitive, with an LD_{50} of 162 mg/kg reported by Gaines. Jones *et al.* (1968) estimated the oral LD_{50} of heptachlor in rats to be 40 mg/kg, and the dermal LD_{50} to be between 200 and 250 mg/kg, as opposed to a dermal LD_{50} of more than 1,600 mg/kg for chlordane. Spynu (1964) reported an acute oral LD_{50} of 210 ± 5 mg/kg for heptachlor in mice. Heptachlor and chlordane were in the minority of pesticides which were more toxic to male than to female rats (Gaines 1969).

Chlordane decreased the toxicity of subsequent parathion treatments, apparently by increasing the levels of serum aliesterases (Williams *et al.* 1967). Chapman and Leibman (1971) pointed out, however, that whereas both DDT and chlordane stimulated the metabolism of parathion to paraoxon and to diethylhydrogen phosphorothionate, only chlordane protected against parathion toxicity. They concluded that factors other than enhanced metabolism must account for the chlordane-parathion interaction.

At levels of two to five mg/kg/day given intraperitoneally for 7 days, chlordane inhibited the uterogenic activity of estrone and decreased the uterine content of estrogen in rats and mice, and at 10-50 mg/kg/day, estrone metabolism was also decreased. Chlordane also increased the levels of estradiol 17β, testosterone, progesterone, and deoxycorticosterone, while heptachlor increased estrone metabolism (Welch *et al.* 1969, 1971). The number of pregnant mice decreased when females were treated intraperitoneally with 25 mg/kg chlordane per week (Welch *et al.* 1971). Mestitzova (1967) found that heptachlor resulted in decreased litter size within and between generations, a decrease in the survival of suckling rats, especially between 24 and 48 hours after birth, and cataracts in survivors. No clinical toxicity was observed when heptachlor levels of six mg/kg were fed for 18 months. In another study, Green (1970) found a decrease in the number of pups born when rats were fed heptachlor levels equal to the maximum food allowances although no increase in the number of abnormal pups was found. In cell culture of human Chang-strain liver cells, chlordane inhibited the replication of both vaccinia and polio virus (Gabliks 1967).

Terracini (1967) observed heptachlor to be weakly carcinogenic in rats, mice, and dogs. In contrast, no effect of long-term feeding of heptachlor was found by Cabral and co-workers (1972) and a tumorstatic effect was claimed for chlordane when urethane was used as the carcinogen (Yamamoto *et al.* 1971). More recent studies demonstrated that mice fed 25 or 50 ppm chlordane had significantly more carcinomas than control mice (Carter 1976, Train 1975). Markaryan (1972) considered several organochlorines, including heptachlor, to be mutagenic with the levels of induced mutations lower than with alkylating agents or irradiation, and consisting mostly of chromatid aberrations. The World Health Organization characterized chlordane as carcinogenic in one species and heptachlor as uncertainly carcinogenic (Vettorazzi 1975).

In summary, these data demonstrate that heptachlor and chlordane exert considerable influence on enzymic and hormonal levels, even at dosages which are not overtly toxic. Although gross congenital abnormalities do not seem a common consequence, chlordane and heptachlor obviously affect reproduction at all stages. Heptachlor and chlordane are carcinogenic, and some evidence for mammalian mutagenicity also exists.

Endosulfan

Endosulfan is the common name for 6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxide, a non-systemic stomach and contact insecticide introduced by Farbwerke Hoechst AG in 1956 under the name Thiodan. For its manufacture, the Diels-Alder adduct of hexachlorocyclopentadiene and cis-1,4-diacetoxybutene is hydrolyzed to the diol which is then reacted with thionyl chloride. Endosulfan is a mixture of two isomers, the *alpha* isomer having a melting point of 108° to 110°C, and the *beta* isomer having a melting point of 208° to 210°C. The technical product is a brownish crystalline solid with an odor of sulfur dioxide and a melting point of 70° to 100°C. It has no measurable vapor pressure at 75°C, has a water solubility less than one ppm, and is moderately soluble in most organic solvents.

Degradation:

Biological--Bacteria and green algae decomposed endosulfan to endosulfan-alcohol (Gorbach and Knauf 1971). Martens (1972) identified the major metabolic products of microbial endosulfan degradation as the sulfate and the endodiol. In soil with natural microbial populations, a maximum of 5.4 percent of the ^{14}C-endosulfan was converted to $^{14}CO_2$ in fifteen weeks; 30 to 60 percent was converted to endosulfan sulfate (Martens 1972). In water, microbial degradation was favored by neutral, aerobic conditions (Greve and Wit 1971). Under aerobic conditions unidentified soil bacteria converted *alpha*-endosulfan as well as *beta*-endosulfan to their respective alcohols, but the ether was obtained only from *beta*-endosulfan (Perscheid et al. 1973).

In mice, ^{14}C-endosulfan was converted to its sulfate prior to tissue storage while fecal excretion included endosulfan itself, endosulfan diol, and endosulfan sulfate. Compounds other than the sulfate were apparently restricted to the liver and kidneys (Deema et al. 1966). In rats and mice, the *alpha* isomer was more rapidly excreted than the *beta* isomer. Five metabolites of endosulfan were detected, including the hydroxyether and the *gamma*-lactone; nevertheless most endosulfan was recovered unaltered (Schuphan et al. 1968). When two ewes were fed 0.3 mg/kg of ^{14}C-endosulfan, the radioactivity was almost totally eliminated within 22 days; fifty percent was eliminated in the feces, 41 percent in the urine, and one percent was excreted in the milk. Milk residues fell to two ppb by the twenty-second day, and to no more than 0.03 ppb after 40 days. Urinary metabolites included 1,4,5,6,7,7-hexachloro-2,3-bis-(hydroxymethyl)-bicyclo-2,2,1-heptene-5 (endosulfan alcohol) and 1-*alpha*-hydroxy endosulfan (Gorbach et al. 1968).

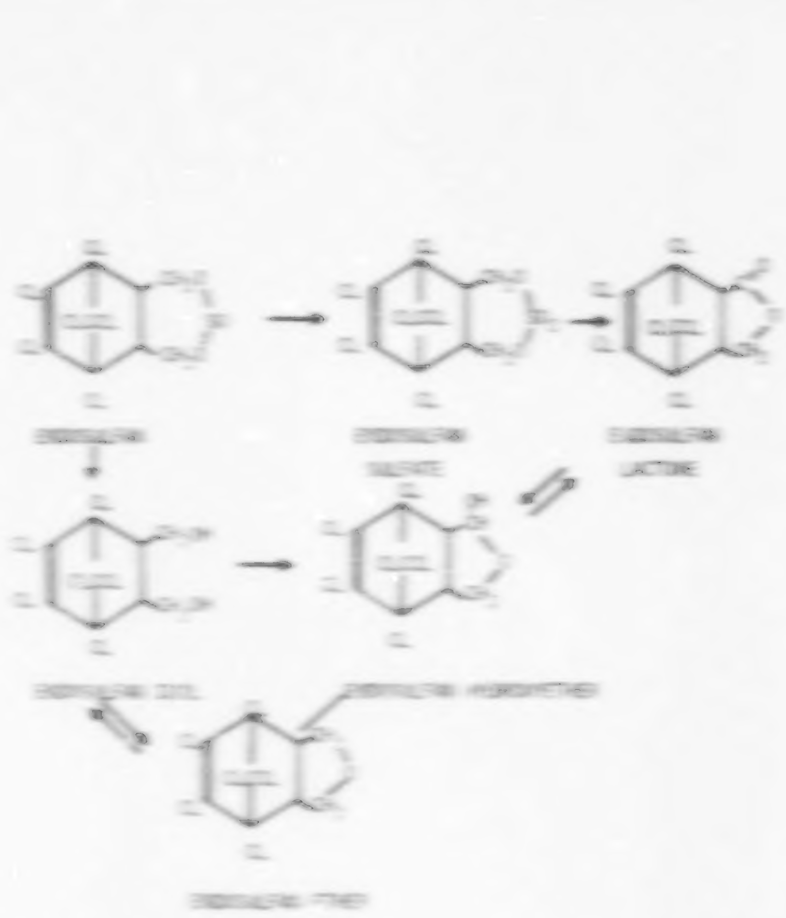
The data on biological degradation of endosulfan are summarized in Table 38 and Figure 16.

Chemical and physical--In neutral solution, the *alpha* and *beta* isomers each decomposed to two unidentified but not identical compounds, while in alkaline solution both isomers formed the diol, which was then open to further degradation (Schuphan and Ballschmiter 1972).

Photolytic--When endosulfan was irradiated by UV light at wavelengths approximating sunlight, the *alpha* isomer was dechlorinated in hexane solution. The *beta* isomer was stable to UV light in hexane, but lost one chlorine if in dioxan-water solution (Schumacher et al. 1971, 1973). Archer and co-workers (1972) obtained endosulfandiol as the major product of UV irradiation. Other photoproducts included the *alpha*-hydroxyether, a lactone, an ether, and some minor unidentified products, but no endosulfan sulfate. No degradation occurred in the dark (Archer et al. 1972). Under laboratory and greenhouse conditions, endosulfan sulfate and endosulfan ether were reported to be the major metabolites (Beard and Ware 1969).

Table 58. BIOLOGICAL DEGRADATION OF ENDOSULFAN.
STRUCTURES OF METABOLITES ARE SHOWN IN FIGURE 15

Organism or conditions	Product	Reference
Molds	endosulfan sulfate and endodiol	Martens *et al.* 1972
Bacteria	endosulfan sulfate and endodiol	Ibid.
Actinomycetes	endosulfan diol	Ibid.
Soil organisms	endosulfan sulfate	Stewart and Cairns 1974
Potatoes	endosulfan sulfate	Ibid.
Mouse	endosulfan sulfate and endosulfan diol	Deema *et al.* 1966
Mouse	endosulfan *gamma*-lactone and endosulfan hydroxyether	Schuphan *et al.* 1968
Rat	endosulfan *gamma*-lactone and endosulfan hydroxyether	Ibid.
Sheep	endosulfan alcohol and *alpha*-hydroxyether	Gorbach *et al.* 1968


ENDOSULFAN
ENDOSULFAN SULFATE
ENDOSULFAN LACTONE
ENDOSULFAN DIOL
ENDOSULFAN HYDROXYETHER
ENDOSULFAN ETHER

METABOLISM OF ENDOSULFAN (AFTER MENZIE, 1974)

Figure 14

Transport:

Within soil--Of 6.7 kg/ha soil-applied endosulfan, 90 percent of the residues were recovered from the top 15 cm of soil, and nine percent from the 15 to 30 cm layer (Stewart and Cairns 1974). When endosulfan was applied to a broad spectrum of soils in columns, water percolation of the soils permitted recovery of 64 percent of the endosulfan from Lakeland sand, but no more than seven percent from sandy clays. Loam recoveries were intermediate (Bowman et al. 1965).

Between soil and water--Endosulfan was found as a pollutant of the Main, Rhine, and Regnitz rivers in Germany (Herzel 1972), with levels of up to 0.70 ppb in the Rhine (Greve and Wit 1971). Richardson and Epstein (1971) concluded that flocculation would not significantly decrease water levels of endosulfan. Greve and Wit (1971) found that endosulfan readily adsorbed to river silt, and that 85 percent could be removed by filtration.

Into organisms--When potato foliage was sprayed eight times with 0.6 kg/ha of endosulfan, both potato peel and pulp contained 0.01 ppm endosulfan (Stewart and Cairns 1974). Rate of penetration of endosulfan and its metabolites into beet and bean plants was of the order: *beta* isomer > sulfate > ether > *alpha* isomer > diol. Only the diol was not transferred to the roots in detectible amounts.

The common mussel, *Mytilus edulis*, reportedly took up *alpha* and *beta*-endosulfan differentially, although the same study suggested that endosulfan was not absorbed directly from seawater by the mussel, but rather was taken up with suspended particles to which it was adsorbed (Roberts 1975). When pigs were fed an acute oral dose of 2 ppm, organ residues after 27, 54, and 81 days were reportedly insignificant compared to organ residues of DDT in pigs fed seven ppm of the latter. Exact residues were not available, however (Maier-Bode 1966).

Volatilization:

Endosulfan volatilized more readily from glass than from leaf surfaces, and more readily from sugar beet foliage than from bean foliage. Under controlled laboratory conditions, endosulfan isomers and metabolites volatilized from glass in the order: *alpha* isomer > ether > *beta* isomer > sulfate > diol but at the higher temperatures of the greenhouse, the order was: *alpha* isomer > ether > *beta* isomer > diol > sulfate (Beard and Ware 1969).

Persistence:

Endosulfan is an infrequently found residue in agricultural soils of southern Ontario (Harris et al. 1966). Stevens et al. (1970) found endosulfan residues in one third of soils with a history of regular pesticide usage (vegetable, cotton, forest), and in nine percent of soils with a history of little or no pesticide use (root vegetables, grains). Residues in vegetable/cotton soils were as high as 1.22 ppm; in forests as high as 4.63 ppm; in grain/root crop soils as high as 0.92 ppm. Mullins et al. (1971) found endosulfan in only one of fifty randomly selected Colorado soils, even though forty-one of the soils contained pesticide residues.

When soil was treated with 6.7 kg/ha of endosulfan, 50 percent of the *alpha* isomer was converted to endosulfan sulfate within 60 days. The *beta* isomer required 800 days for 50 percent conversion to endosulfan sulfate (Stewart and Cairns 1974). The sulfate was characterized by the authors as being equally toxic to insects and "relatively stable", but no persistence times for it were given. Residues of endosulfan sulfate, (0.3 ppm), *beta*-endosulfan (0.06 ppm) and *alpha*-endosulfan (0.01 ppm) were found in potato peels, and 0.03 ppm endosulfan sulfate in the potato pulp. The authors concluded that conversion to endosulfan sulfate occurred in the potato (Stewart and Cairns 1974).

When one lb/A (0.89 kg/ha) of granular endosulfan was applied to Bermuda grass, 13.7 percent remained after 96 days, during which two crops of hay were cut and 16.5 inches of rain fell (Byers et al. 1965). In flooded rice fields of east Java, agricultural levels of endosulfan (1.4 liter of Thiodan 35 EC in 750 liters of water per hectare) resulted in elevated endosulfan levels for no more than five days. Maximum levels in mud were 1.9 ppm (Gorbach et al. 1971). Greve and Verschuuren (1971) reported endosulfan residues of up to 0.07 ppb in river water, and up to 0.01 ppb in drinking water. Greve and Wit (1971) estimated a half-life of five weeks for endosulfan in water at pH 7, but of five months at pH 5.

Effects on Non-Target Species:

Microorganisms--Gorbach and Knauf (1971) did not find any inhibition of bacterial action by endosulfan under field conditions. Knauf and Schulze (1973), however, noted that the aquatic microorganism *Chlorella vulgaris* responded to 0.3 ppm of endosulfan by reducing its rate of reproduction.

Invertebrates--Luedemann and Neumann (1962) studied the effects of endosulfan on ten aquatic invertebrates. Mussels (*Dreissena*) were unaffected by less than ten ppm and killed by 100 ppm endosulfan while the American river crab (*Cambarus affinis*) was affected by 0.1 ppm and killed by one ppm. Most sensitive was the flea crab (*Carinogammarus*), which was killed by 0.005 ppm, with toxic effects noted at 0.001 ppm.

Fish and amphibians--Endosulfan is highly toxic to fish. Gorbach and Knauf (1971) considered the lethal levels to be between 0.001 and 0.01 ppm, depending on the species. Toor et al. (1973) found 0.007 ppm to be the threshold level of endosulfan toxicity in carp, while 0.005 ppm was the maximum sublethal dose. Rainbow trout fry were adversely affected by 0.006 ppm and pike by 0.001 ppm endosulfan, while the lethal levels were 0.01 ppm and 0.005 ppm for the two species, respectively (Luedemann and Neumann 1962). Greve and Wit (1971) considered one ppb to be a sufficiently great concentration of endosulfan to cause large fish kills under unfavorable conditions. Schoettger (1970) reported

TL$_{50}$ levels of endosulfan to be as low as 0.3 ppb in trout, but noted that trout eggs could tolerate 50 ppm for two hours. Endosulfan at one lb/A (1.12 kg/ha) was toxic to the toad *Bufo boreas* (Mulla et al. 1963).

Birds--The LD$_{50}$ of endosulfan for starlings was 35 mg/kg (Schafer 1972); for mallards, 200 to 705 mg/kg, and for ring-necked pheasants, 620 to 1000 mg/kg (Martin 1968). In tests on chicken and quail embryos, neither immersion of the eggs nor injection of the embryos with endosulfan caused birth defects, but sterility occurred in both males and females so treated (Lutz-Ostertag and Kantelip 1971).

Mammals--The LD$_{50}$ of endosulfan to rats was 100 mg/kg (Schafer 1972). In mice, the acute oral LD$_{10}$ was estimated at 13 mg/kg in highly inbred (Balb/c) mice; chronic dietary intake of ten ppm was sublethal (Deema et al. 1966). Diet was found to affect tolerance to endosulfan in rats, which had an acute oral LD$_{50}$ of 5.1 ± 1.5 mg/kg endosulfan if fed a protein-deficient diet; when casein constituted 81 percent of the diet, the LD$_{50}$ rose to 98 ± 7 mg/kg endosulfan (Boyd et al. 1970). Endosulfan was the only chlorinated hydrocarbon insecticide found not to be accumulated in mammals (Vettorazzi 1975).

Toxaphene

Toxaphene is the name for a mixture of chlorinated camphene compounds of uncertain identity, with a chlorine content of 67 to 69 percent by weight. It was introduced by Hercules Powder, Inc. in 1948 and is used heavily on cotton, formerly with DDT and now with methyl parathion or parathion. Other names for toxaphene include polychlorocamphene (USSR), chlorinated camphene, and octachlorocamphene. It is similar in composition and origin to several other chlorinated terpenes, such as strobane. Toxaphene is the most widely used chlorinated hydrocarbon insecticide (Matsumura *et al.* 1975).

Toxaphene is a yellow waxy solid with a mild terpene odor which softens between 70° and 90°C. It is noncorrosive in the absence of moisture, and its solubility in water is 0.4 ppm (La Fleur 1974, Metcalf and Sanborn 1975). It is readily soluble in organic solvents, including petroleum oils, and can be dehydrochlorinated by heat, strong sunlight, and by certain catalysts such as iron (Martin 1968).

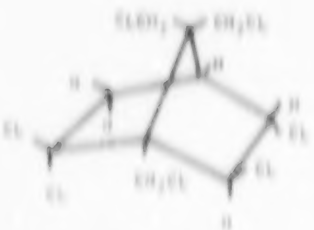
Bevenue and Beckman (1966) considered gas chromatography to be an unreliable method of identifying toxaphene if other pesticides were present. The alternatives, total chloride determination and bioassay, are hardly specific to toxaphene, and so are limited to situations in which the history of contamination is known (Guyer *et al.* 1971). Kawano *et al.* (1969) reported that a mixture of sulfuric-fuming nitric acid can be used to remove parathion and DDT from samples before analyses for toxaphene.

Despite prolonged and heavy use, relatively little is known of the action or fate of toxaphene in the environment, since the complexity of its composition makes residue analyses tedious and uncertain (Guyer *et al.* 1971). Nelson and Matsumura (1975) circumvented the analytical difficulties by constructing a simplified toxaphene, consisting of a few toxaphene components, but this approach is restricted to laboratory experiments. Isolation of some major components of toxaphene was achieved recently by Matsumura *et al.* (1975) who elucidated the structure of two toxaphene components which were said to account for up to 56 percent of the mixture's toxicity to mammals. Casida and his co-workers established that toxaphene consists of at least 175 polychlorinated derivatives of camphene, one of which was identified as 2,2,5-*endo*,6-*exo*,8,9,10-heptachlorobornane, shown in Figure 16 (Casida *et al.* 1974, Holmstead *et al.* 1974, Palmer *et al.* 1975). An octachloronorbornane constituting six percent of the mixture was isolated and was found to be fourteen times as toxic to mammals as toxaphene (Khalifa *et al.* 1974). Anagnostopoulos, Parlar, and Korte (1974) also isolated several toxaphene components, of which three are shown in Figure 15. Together these constituted five to eight percent of technical toxaphene.

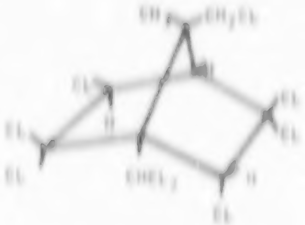
Degradation:

Biological--rats fed 20 mg/kg ^{36}Cl-toxaphene excreted about 37 percent of the radioactivity unchanged in the feces, but 15 percent was excreted in the urine, mostly as ionic chloride. Repeated doses led to decreases in the fecal, or unmetabolized, fraction (Crowder and Dindal 1974). Ohsawa *et al.* (1975) found half the radioactivity in the urine as chlorides, and concluded that biodegradability was characteristic of all toxaphene fractions, but that relatively few of the fractions were toxic to the rat.

In dairy cattle, lactating cows fed up to 20 ppm toxaphene for 77 days excreted it in their milk for only fourteen days, after the end of the treatment, but a single cow who was nearly dry excreted toxaphene in her milk for longer periods. The authors concluded that excretion in milk was a major route of toxaphene elimination in cattle (Zweig *et al.* 1963). There were no data available on the degradation of toxaphene by microorganisms, plants or invertebrates.


ClCH2
CH2Cl
H
H
H
Cl
Cl
Cl
CH2Cl
Cl
H
Cl

2,2,5-ENDO,6-EXO,8,9,10-HEPTA-
CHLOROBORNANE (CASIDA ET AL. 1974)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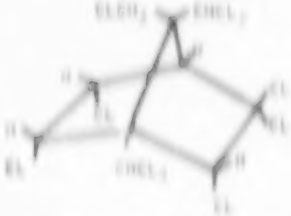
CHCl2
H
Cl

(ANAGNOSTOPOULOS ET AL. 1974)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CHCl2
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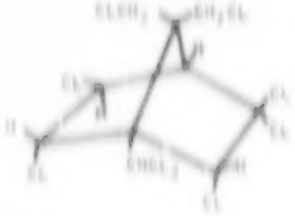
Cl

(ANAGNOSTOPOULOS ET AL. 1974)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CH2Cl
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CHCl2
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Cl

(ANAGNOSTOPOULOS ET AL. 1974)

STRUCTURES OF TOXAPHENE COMPONENTS

Figure 15

Chemical and physical--Clay-catalyzed decomposition of toxaphene is purportedly mediated by acidic diluents of the clays and increasing temperature as well as increasing acidity speeded the degradation (Fowker *et al.* 1960). Physical degradation of toxaphene has not been reported on. Inasmuch as toxaphene consists of mixed chlorinated camphenes it seem plausible that degradative methods safe for DDT and cyclodiene insecticides would be safe for use with toxaphene. Iron is known to catalyze dehydrochlorination of toxaphene (Martin 1968).

Transport:

Studies on transport are difficult because of the complexity of the mixture and the difficulty of identifying small quantities of the multitudinous components. Bradley and co-workers (1972) recovered less than one percent of the toxaphene applied to cotton plots in the surface runoff water; of the toxaphene lost to water flow, 75 percent was associated with sediment. After ten years, up to 95 percent of the toxaphene residues remaining in Houston black clay were in the top 12 inches of soil (Swoboda *et al.* 1971). Toxaphene applied to Dunbar soil in a South Carolina field plot was found in ground water within two months however. The half-life in soil was estimated to be 120 days, but groundwater contamination persisted for the entire year of observation (La Fleur *et al.* 1972). Toxaphene is also a contaminant of the lower Mississippi and its tributaries (Barthel *et al.* 1969). Furthermore, traces of two heptachloronorbornenes which could be degradation products of toxaphene have been found in New Orleans drinking water at levels of 0.04 to 0.06 ppb (ANON. 1974). Klein and Link (1967) found that when toxaphene was sprayed on kale, most of the pesticide disappeared within three days, even in the absence of rain; after seven days and two inches of rain, only eight percent remained on the leaves and after 28 days, only traces were found.

Veith and Lee (1971) observed that toxaphene residues in the top five centimeters of lake sediment increased for 190 days following treatment;

and then decreased by a factor of two every 120 days, apparently by transport to deeper strata; a rate of movement of 0.4 to 1.1 centimeters per day was estimated. Toxaphene was not, however, detected below 20 centimeters. Lake water did not leach toxaphene out of sediment under laboratory conditions.

When a toxaphene-contaminated stream was dredged, toxaphene residues increased in estuarine fauna and flora. Oysters (*Crassostrea virginica*) contained two to five ppm, but mummichogs (*Fundulus heteroclitus*) contained more than 200 ppm toxaphene. Even marsh grass (*Spartina alterniflora*) contained 7.5 ppm toxaphene, providing a rare example of transport of toxaphene from soil into plants. The authors postulated that uptake by the marsh grass was facilitated by the species' ability to transport and accumulate salt (Reimold and Durant 1974). Adsorption on charcoal decreased the toxicity of toxaphene to goldfish (Barron 1969).

When toxaphene was studied in an aquatic terrestrial ecosystem (Metcalf et al. 1971), algae, snails, and mosquitofish were found to contain 6902-fold, 9600-fold, 890-fold and 4247-fold as much ^{14}C-toxaphene, respectively, as was found in the water, indicating that toxaphene is highly accumulative in aquatic food chains (Sanborn and Metcalf 1975).

Persistence:

After 16 years, residues of toxaphene in Congaree sandy loam were estimated at 49 percent (Nash and Harris 1973). After 14 years under conditions predisposing to maximum persistence, 45 percent remained in the soil (Nash and Woolson 1967). Randolph et al. (1960), however, observed a loss of 50 percent of toxaphene in one year in Texas soils. The data in these studies included DDT residues similar to those reported for toxaphene.

When toxaphene was applied for five years to a California soil, the total application reached 103.6 lbs/A (116 kg/ha), with soil residues of ten to 15 ppm after the fifth application. Four years later, four to six ppm remained, a loss of approximately 60 percent over four years (Hermanson et al. 1971).

Persistence of toxaphene in lakes was found to depend heavily on the nature of the lake: shallow eutrophic lakes were successfully restocked with fish after one year, whereas deep, oligotrophic lakes were still toxic after five years (Terriere et al. 1966). Levels of toxaphene were 88 ppb in treatment of the shallow lake: one year later, water levels had fallen to one ppb, but aquatic plants contained 500 ppb, aquatic animals (other than fish) 1,000 to 2,000 ppb, and trout contained up to 20,000 ppb. Similar levels were observed in trout from the deep lake with sparse biota (Terriere et al. 1966). Similar results were obtained by Johnson et al. (1966), who concluded that sorption rather than degradation of toxaphene was responsible for the detoxification of toxaphene in eight Wisconsin lakes.

Effects on Non-Target Species:

Microorganisms--When pure cultures of marine phytoplankton were grown in the presence of pesticides, toxaphene at 0.15 ppm was absolutely toxic, preventing growth in all species tested. Even 0.01 ppm depressed growth in all species except the rather resistant *Protococcus;* 0.00015 ppm prevented all growth of *Monochrysis lutheri*, the most sensitive species in the group and 0.000015 ppm toxaphene still caused 78 percent growth inhibition in this species (Ukeles 1962). At high agricultural levels of application, toxaphene was reported to have significant effect on the numbers of bacteria or fungi in soil within ten months of five annual applications (Martin et al. 1959). Smith and Wenzel (1947) had reported stimulation of fungi and bacteria which were able to use toxaphene for growth.

Plants--Cullinan (1949) observed degradation of toxaphene during tests of phytotoxicity and soil effects. Dias-Mena (1956) also observed phytotoxicity with toxaphene, as with all chlorinated hydrocarbons, under his experimental conditions. Toxaphene reportedly inhibited photosynthesis in alfalfa unless rain occurred within six days. Increasing temperature decreased the degree of inhibition (Kralovic 1976).

Invertebrates--Oysters (*Crassostrea virginica*) exposed to one ppb/liter each of DDT, toxaphene, and parathion gained less wieght and were more subject to mycelial fungal infections than control oysters, while other single pesticides at one ppb did not have any discernible effect (Lowe et al. 1970). Hard shell clams (*Mercenaria mercenaria*) and oysters (*Crassostrea virginica*) were more affected by toxaphene during the embryonic than larval stage (Davis and Hidu 1969).

Fish and amphibians--Toxaphene is among the most toxic of pesticides to fish; with only rotenone and endrin acknowledged to be more toxic (Adlung 1957). Little species difference was observed in the relative sensitivities of fish families to toxaphene: comparisons were made between *Ictaluridae*, *Cyprinidae*, *Centrarchidae*, and *Salmonidae* (Macek and McAllister 1970). Toxaphene was more toxic to mosquitofish in static than in flowing solutions (Burke and Ferguson 1969). Toxaphene accumulation was found in bluegills (*Lepomis macrochirus*) after treating lakes for rough fish control even though the restocked bluegills grew well (Hughes and Lee 1973).

In fathead minnows (*Pimephales promelas*), water levels of 55 to 1,230 ng/liter (0.055 ppb to 1.23 ppb) resulted in growth disturbance after 90 and within 150 days; the minnows' collagen content was shown to decrease, both absolutely and in relation to calcium content, and the increased mineralization was expressed by increased fragility. Under stress, toxaphene-treated minnows' backbones fractured (Mehrle and Mayer 1975a). Brook trout (*Salvelinus fontinalis*) were exposed to toxaphene from 22 days before to ninety days after hatching. Although hatching itself was not affected, growth was significantly decreased by 139 and 288 ng/liter (0.139 to 0.288 ppb) within 30 days, and by 39 ng/liter (0.039 ppb) within ninety days after hatching. Whole-body collagen was decreased within 15 days of hatching, and calcium and phosphorus content increased (Mehrle and Mayer 1975b).

Toxaphene was less toxic to anuran amphibians than were endrin, aldrin, or dieldrin, and had approximately the same toxicity as DDT (Ferguson and Gilbert 1967). Tolerance of up to 200-fold was found in amphibians near insecticide treated cotton fields (ibid).

Birds--The LD_{50} of toxaphene in seven-day old mallard ducklings was 30.8 mg/kg, with 95 percent confidence limits of 23.3 to 40.6 mg/kg (Tucker and Crabtree 1970). Mallards were less sensitive at three to five months of age, with an LD_{50} of 70.7 mg/kg and 95 percent confidence limits of 37.6 to 133 mg/kg. Mature sharp-tailed grouse had an LD_{50} of 10-20 mg/kg. Symptoms were seen within 20 minutes in some species, but mortality generally occurred between two and fourteen days (Tucker and Crabtree 1970).

Limited tests with young chickens suggested that toxaphene, in contrast to *o,p'*-DDT and *p,p'*-DDE, decreased sleeping time (Stickel 1973). Toxaphene increased the thyroid growth of bobwhite quail when birds were treated with 50 ppm for three or four months while liver weights did not increase even after 500 ppm were fed for the same length of time (Hurst et al. 1974).

Mammals--The acute oral LD_{50} of toxaphene in rats is generally considered to be 90 mg/kg in males and 80 mg/kg in females (Servintuna 1963, Guyer et al. 1971) although Jones et al. (1968) claimed an LD_{50} of 283 mg/kg. The dermal LD_{50} was slightly over 1,000 mg/kg (Servintuna 1963, Jones et al. 1968). Schindler (1956) found that toxaphene controlled voles when applied at two kg/ha, and Lange and Crueger (1960) found that four liters/ha of a 50 percent toxaphene solution resulted in a thorough kill of field mice.

In rats, less than additive effects were reported for the combination of carbofenthion, diazinon, or parathion with toxaphene, but carbaryl or malathion interacted additively (Keplinger and Deichmann 1967). The mode of action of toxaphene poisoning is not understood, but it is thought to be pharmacologically similar to that of the cyclodienes, since cyclodiene resistance invariably confers toxaphene resistance in insects (Brooks 1974, Vol. 2). No effects of toxaphene on rat reproduction were found during a multigeneration study (Kennedy *et al.* 1973).

Conclusions

Although data on organochlorine insecticides are by no means complete, it is apparent that, in less than forty years, they have become almost universal contaminants of land, water, and living things. Of the 17 compounds for which a literature search was carried out, only methoxychlor is biodegradable. With respect to the other compounds, it must be assumed:

1. that, over the period of their persistence, all will move through soil, into water, into the atmosphere, and into living organisms;
2. that there are no efficient biological pathways for their degradation to naturally occurring substances;
3. that the effects of some, and perhaps all, of these compounds on microorganisms, fish, birds, and even man may eventually prove as disastrous as they seemed benign originally.

Accordingly, it is recommended that waste chlorinated hydrocarbon insecticides be burned or chemically degraded, with suitable precautions for disposal of chlorine and other toxic gases. Disposal in soil, whether in shallow pits or deep wells, is considered utterly undesirable, since the available evidence is that dispersal would precede any significant degradation. The sole exception to these conclusions is methoxychlor, which might be disposed of in flooded soil or in sequestered ponds, provided adequate precautions were taken against contaminating natural waters. Endosulfan is not excepted from the conclusion against soil or water disposal, since the few available data suggest that its major terminal residue endosulfan sulfate, which shares most of the toxic properties of endosulfan itself, is very persistent in soil.

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ORGANOPHOSPHORUS INSECTICIDES

Methyl Parathion and Parathion

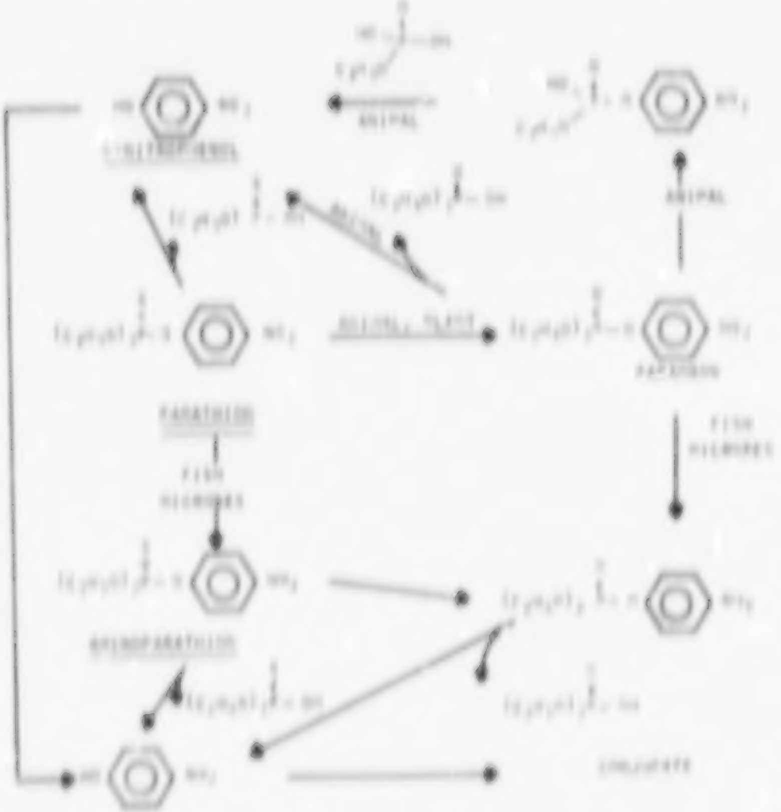
Methyl parathion is the common name for *O,O*-dimethyl *O-p*-nitrophenyl phosphorothioate, known as parathion-methyl in western Europe and as metaphos in the USSR. Introduced in 1949 by Farbenfabriken Bayer as "Dalf" or "Folidol-M", methyl parathion is a nonsystemic contact and stomach insecticide. When pure, it is a white crystalline powder with a melting point of 35° to 36°C and a vapor pressure of 0.97×10^{-5} mm mercury at 20°C. At 25°C its solubility in water is 55 to 60 ppm; it is slightly soluble in light petroleum and mineral oils and soluble in most other organic solvents. The technical product is a light to dark tan liquid of about 80 percent purity (Martin 1968).

Parathion is the common name for *O,O*-diethyl *O-p*-nitrophenyl phosphorothioate, also a nonsystemic contact and stomach insecticide. Known as thiophos in the USSR, parathion is a pale yellow liquid with a boiling point of 157°C at 0.6 mm mercury, and a vapor pressure of 3.78×10^{-5} mm mercury at 20°C. Its solubility in water is 24 ppm at 25°C, and it is slightly soluble in petroleum oils. It is miscible with most organic solvents. Parathion can be hydrolyzed in alkaline media, but only one percent is hydrolyzed in 62 days at pH five or six (Martin 1968).

Parathion is synthesized by condensing diethyl phosphorochloridothioate with sodium *p*-nitrophenate, while methyl parathion requires dimethyl phosphorochlorodithioate.

Degradation:

Biological--Biological pathways of parathion degradation are shown in Figure 16; pathways for methyl parathion are analogous. The metabolic products of microbial degradation of methyl parathion are listed in Table 59, as are data representative of mammalian degradation/metabolism.


4-NITROPHENOL
ANIMAL
PARATHION
ANIMALS, PLANTS
PARAOXON
FISH
MICROBES
AMINOPARATHION
CONJUGATE

DEGRADATION PATHWAYS FOR PARATHION (MODIFIED FROM [illegible] AND [illegible] (1975). DEGRADATION PATHWAYS FOR METHYL PARATHION ARE ANALOGOUS.

Figure 16

Table 59. BIODEGRADATION OF METHYL PARATHION

Organism	Products	Reference
Pseudomonas	unidentified	Neumann 1967
Mouse	dimethyl phosphoric acid dimethyl phosphorothioic acid methyl phosphoric acid methyl phosphate, phosphate	Hollingsworth *et al*. 1967

The microbial degradation of parathion has been documented repeatedly (Singh 1974, Boush and Matsumura 1967, Griffiths and Walker 1970, Getzin and Rosefield 1968). Table 60 lists specific organisms and the products of their degradation.

Naumann (1959) observed that methyl parathion was 99.9 percent degraded in three weeks if 0.5 to one percent (5,000 to 10,000 ppm) were added to the soil. Sterilized soil had first to be recolonized before degradation took place. Sethunathan (1973a, 1973b) and Sethunathan and Yoshida (1973a, 1973b) found that parathion was degraded more rapidly in flooded soil or under anaerobic conditions than under aerobic conditions. If flooded soil was additionally inoculated with *Flavobacterium*, the half-life of parathion was approximately 0.76 days, compared to 3.5 days in uninoculated soil. Aminoparathion was the primary anaerobic metabolite, with *p*-nitrophenol as the terminal residue. *Bacillus* species were found which were capable of degrading *p*-nitrophenol to carbon dioxide. Maximum initial degradation of parathion occurred in acid sulfate soil under anaerobic conditions and repeated applications enhanced the degradation in alluvial soils. Ring cleavage and CO_2 production, however, occurred only under aerobic conditions.

Penicillium waksmani degraded up to 1000 mg/ml (1000 ppm) parathion on agar, but was inhibited at higher levels of parathion. After 15 days, 61 percent of the parathion had been degraded, with aminoparathion as the major product (Rao and Sethunathan 1974). The addition of organic matter shifted degradation from the hydrolytic pathways typical of aerobic unflooded soils to reductive pathways, increasing production of aminoparathion (Rajaram and Sethunathan 1975). A mixed bacterial culture degraded a parathion-xylene formulation in aqueous medium at a maximum rate of 50 mg/liter/hour, or with an optimum parathion concentration of 5000 mg/liter (5000 ppm) in batch cultures. Both the xylene diluent and the parathion were metabolized, the latter preferentially

Table 60. BIODEGRADATION OF PARATHION

Organism	Products	Reference
Clarias nigrutus	unidentified	Singh 1974
Chlorella sp.	aminoparathion and p-nitrophenol	Mackiewicz *et al.* 1969
C. vulgaris	unidentified	Palut and Syrowatka 1968
C. pyrenoidosa	aminoparathion and 2 unknowns	Zuckerman *et al.* 1970
C. protuous	aminoparathion and 3 unknowns	Ibid.
Flavobacterium	p-nitrophenol	Sethunathan 1973b
Flavobacterium	hydrolysates, p-nitrophenol	Sethunathan and Yoshida 1973
Bacillus	NO_2	Sethunathan 1973a
Bacillus sp.	p-nitrophenol	Siddaramappa and Sethunathan 1970
Pseudomonas sp.	NO_2	Ibid.
Pseudomonas	unidentified	Naumann 1967
Trichoderma viride	22.2% water soluble	Matsumura and Boush 1968
Pseudomonas melophthora		Boush and Matsumura 1967
Penicillium waksmani	aminoparathion	Rao and Sethunathan 1974
Cow	p-aminophenyl glucuronide	Pankaskie *et al.* 1952
Rat liver	paraoxon, p-nitrophenol diethyl hydrogenphosphate diethyl hydrogenphosphorothionate	Neal 1967

at slightly alkaline pH. Complete air saturation, a pH between 7.0 and 7.5, a temperature of 37°C were optimal. Five of six pseudomonads isolated from the mixed culture used an ortho ring fission system for *p*-nitrophenol metabolism while the sixth did not grow on *p*-nitrophenol (Munnecke and Hsieh 1975).

Naumann (1967) found a *Pseudomonas* species which was able to dissolve crystalline methyl parathion from the medium in four to eight weeks. Siddaramappa *et al.* (1973) found that *Pseudomonas* could degrade parathion (formulated as Folidol) in a 1,000 ppm aqueous solution with 20 grams of alluvial soil and 24 ml water; two weeks were required for the degradation. In sewage lagoons, parathion was degraded less rapidly than malathion but more rapidly than diazinon (Halvorson *et al.* 1971), and both parathion and methyl parathion were inactivated within days by polluted stream water (Yasuno et al. 1966). *Bacillus subtilis* was highly effective in converting parathion to aminoparathion and methyl parathion to methylaminoparathion (ibid).

In summary, both parathion and methyl parathion appear to be eminently degradable compounds in that microorganisms capable of degrading them exist. Since both compounds appear to serve as energy sources for the degrading microorganisms, selection for more efficient degradation is plausible, in contrast to the situation found for cometabolism by organochlorine-degrading microorganisms (Alexander 1972).

On plants, evaporation and photolysis predominated as modes of parathion removal (Popov and Khol'kin 1958). Paraoxon decomposed more rapidly than parathion on citrus foliage (Spear *et al.* 1975). The major metabolites of methyl parathion in rice grains were thiophosphoric acid and *O*,*O*-dimethyl thiophosphoric acid (Tomizawa et al. 1962).

Parathion is rapidly metabolized in mammals. In rabbits, the maximum blood levels were found twenty minutes after intravenous injection of six to ten mg/kg parathion, and removal from the blood occurred in 30 to 60 minutes. A second injection was cleared less rapidly (Gar *et al.* 1955). In rats, maximum levels of ^{32}P were found in liver, brain, lungs, and kidneys within two hours after ingestion; bone levels of ^{32}P increased for only seventy-two hours (Kortus *et al.* 1968).

Hollingworth *et al.* (1967a, 1967b) compared the metabolism of methyl parathion with the less toxic fenitrothion *O,O*-dimethyl *O*-(3-methyl-4-nitrophenyl) phosphorothioate. Analysis of urinary and fecal metabolites of the two compounds in mice demonstrated that direct excretion of their oxygen analogs was a significant detoxification mechanism. The parent compounds were not, however, excreted directly. Neither ring hydroxylation nor reduction of the nitro group to the amino group were significant in mice. As dosage increased, the excretion of dimethyl phosphoric acid did not rise proportionally, whereas the levels of desmethyl phosphorothionate increased from a secondary metabolite to the major urinary metabolite. It was concluded that, whereas hydrolytic removal of dimethyl phosphoric acid was the primary murine detoxification mechanism, desmethylation was a secondary route available when levels of the toxicants became too high and the hydrolyzing enzyme systems were overwhelmed.

In rat liver slices, parathion activation to paraoxon was associated with the microsomal or mitochondrial fraction (Nakatsugawa and Dahm 1967, Fukami and Shishido 1963). In female rats, 4.4 percent of the LD_{50} dose was converted to paraoxon within one hour, with 64 percent of the conversion occurring in the liver (Kubistova 1959). In dairy cows, 14 ppm for 61 days produced no detectable parathion in milk, blood, or urine of the cattle; conjugation with a *p*-aminoglucuronide prior to urinary excretion was postulated (Pankaskie *et al.* 1952). Gyrisco and his co-workers (1959) found no residues in the milk of four dary cows fed ten ppm of parathion for three years. In sheep, however, 0.10 to 0.15 percent of a daily ingested dose of 2.0 to 5.5 mg/kg was excreted in the milk; four to five mg/kg parathion also decreased the volume of milk produced (Alamanni and Cossedu 1969).

Photolytic--Ultraviolet light between 1,850 and 4,000 Å converts parathion to paraoxon (Payton 1953). On leaf surfaces and glass, sunlight catalyzed the conversion of parathion to paraoxon (about one percent) and to *p*-nitrophenol (El-Refai and Hopkins 1966). Parathion in ethanol is photolyzed to *O,S*-triethyl thiophosphate as a major product, and paraoxon and *O,O,O*-triethyl phosphate as minor products (Grunwell and Erickson 1973). Koivistoinen and Merilainen (1962a, 1962b) claimed that paraoxon was not affected by ultraviolet light. Baker and Applegate (1970) examined the effects of temperature and ultraviolet light on methyl parathion and found that UV radiation increased removal of methyl paration from soils by ten to 14 percent at 30°C and by 14 to 17 percent at 50°C.

Chemical and physical--Parathion is most readily degraded by hydrolysis, which is easily catalyzed by peroxidases or by ferrous iron and EDTA (ethylenediaminetetraacetic acid) (Knaak et al. 1962). In natural aqueous medium approximately 70 percent of the parathion was hydrolyzed in four weeks and 72 percent in six weeks. In another study, 74 percent of the methyl parathion was hydrolyzed in six weeks (Cowart et al. 1971). Hydrolytic degradation eventually proceeded to the terminal residue *p*-nitrophenol, itself toxic.

Hydrolysis of parathion proceeded more rapidly in calcareous sediment than in slightly acid sediments; aminoparathion was the major metabolite (Graetz et al. 1970). At a pH of 7.4 at 20°C, parathion was estimated to have a half-life of 108 days if chemical hydrolysis were the means of degradation; under the same conditions, paraoxon had a half-life of 144 days (Faust and Gomaa 1972). In bottled river water both methyl parathion and parathion were completely degraded within eight weeks (Eichelberger and Lichtenberg 1971). Menzie (1972) considered parathion to be nonpersistent in water.

Under shelf storage, less than four percent of a 50 percent methyl parathion emulsion decomposed in sixteen months (Raman and Krishnamoorthy 1973). Dust formulations of methyl parathion decomposed to give *O*,*O*-dimethylphosphate, its *S*-methyl isomer, *O*-methyl *S*-methyl *O*-*p*-nitrophenylthiophosphate and *p*-nitrophenol (Gota et al. 1960). Parathion could be removed from noncombustible containers by washing, and disappeared from 50 percent ethanol washes within five hours. Sodium hydroxide and sodium hypochlorite were less effective (Hsieh et al. 1974).

Parathion can be converted to paraoxon by seven percent chlorination of water or by one to two ppm of ozone, but potassium permanganate at ten to 40 ppm was not effective (Robeck et al. 1965). The toxic degradation product *p*-nitrophenol, if present to one part per hundred in water, could be removed by the addition of 40 kilograms of sodium hydroxide plus 50 kilograms of bleaching powder for each 200 liters of water: after two to three hours at 100°C, all organic compounds were removed from the water and 320 grams of inorganic salts were produced per liter of water (Mel'nikov et al. 1958).

Radiolysis was successfully used to degrade parathion: 0.1 to 5.0 Mrads resulted in 27 percent degradation of parathion in hexane solution (Lippold et al. 1969) and 10,000 rads of *gamma*-radiation from a ^{60}Co source resulted in better than 80 percent destruction of sufficiently dilute aqueous solutions (Sunada 1967). Sufficiently dilute was defined as concentrations measured in parts per trillion.

Transport:

Within soil--McCarty and King (1966) stated that methyl parathion and parathion migrated rapidly in soil-water systems, but were also degraded rapidly. Lichtenstein et al. (1967) found, on the contrary, that parathion moved through soil less rapidly than aldrin. Some parathion entered water through sorption to loam and transport with loam particles, and one ppm of parathion in the presence of percolating water resulted in small water residues of the pesticide. In soil columns of Nacodoches clay subsoil, parathion leached to 60 inches when

230 inches of rainfall were simulated, while in Houston black clay, 1,725 inches of simulated rain were required to produce leaching to 60 inches (Swoboda and Thomas 1968). Adsorption was found to be the main factor retarding mobility, and Swoboda and Thomas (1968) considered parathion to be dissolving as a liquid phase in an organic fraction of the soil. Parathion applied at 0.1 pounds per acre, followed by flooding and a subsequent application of 0.2 pounds per acre, was degraded before reaching drainage tiles six feet below the soil surface (Johnson *et al*. 1967).

Stewart *et al*. (1971) reported that little leaching occurred in sixteen years after four annual applications of 31.4 pounds of parathion, despite 42 inches of precipitation per year. Wolfe *et al*. (1973) reported that very little parathion was found below the nine inch level six years after 30,000 to 95,000 ppm of parathion were applied to soil. In a 15 year study of pesticide residues in light sandy soil, Voerman and Besemer found that dieldrin and DDT, but not lindane or parathion, leached to 60 centimeters; parathion was not found below 20 centimeters. Hoi *et al*. (1972) did not find parathion much below 2.5 centimeters even with 220 mm of rain during the eight month persistence of parathion under their experimental conditions. Burkhardt and Fairchild (1967) applied parathion (formulated as Niran) in several sandy soils, and observed increased mobility at higher levels of moisture. Racher and co-workers (1972) observed little movement of parathion in charcoal or kaolinite formulations. Parathion contamination of a farm pond and its sediment was correlated with the degree of erosion, suggesting that parathion moved with soil particles rather than by leaching (Nicholson *et al*. 1962).

No mention is made in these studies of the fate or mobility of the degradation products of parathion or methyl parathion.

Between soil and water--When soil was added to parathion in water, 63 percent of the parathion was adsorbed by the soil from a low volume of water (100 ml), but only 35 percent was adsorbed from a larger volume (250 ml). The carrier, the depth of the water, its agitation, and the rate of parathion application all affectud the rate of loss of parathion to soil (Weidhaas *et al*. 1961). King *et al*. (1969) tested the ability of several adsorbents to remove parathion from water, and found that soil with a high clay content was more effective than sandy soil; coal was 2.5 times as effective as soil, and algal systems were ten times as effective as soil. Activated charcoal had 1,000 times the sorptive capacity of soil. Movement of parathion into water from soil appeared to occur by erosion rather than by leaching (Nicholson *et al*. 1962).

Into organisms--The high toxicity of parathion and methyl parathion in most animals makes bioaccumulation unlikely, nor has evidence for it been found in cattle (Pankaskie *et al*. 1952, Cyriaco *et al*. 1959), sheep (Alamanni and Cossedu 1969) or rabbits (Kortus *et al*. 1968).

Methyl parathion was taken up by carrots from soil and retained for up to four weeks (Engst *et al*. 1966a, 1966b). No methyl paraoxon was found, suggesting very slow metabolism (1966a) but five phosphorus-containing degradation products were found, including monomethyl phosphate and dimethyl phosphate (1966b). El-Refai and Hopkins (1966) documented the transfer of ten percent of foliar applications of parathion into the bean plant. Smith (1950) said that only minute amounts of the parathion absorbed from foliar applications was translocated within the plant, and Zuckerman *et al*. (1966) did not find any transfer to roots or soil from foliar applications, but near-insecticidal levels of parathion were transported from soil to leaves.

Bean plants grown in soil containing parathion contained parathion metabolites if and only if microorganisms capable of metabolizing parathion were present in the soil; in sterile root cultures, bean plants translocated only unaltered parathion (Mackiewicz *et al*. 1969).

Studies of parathion movement in a model cranberry bog demonstrated that both fish and microorganisms are capable of accumulating parathion; fish metabolized the compound better than did mussels, and dead fish accumulated 80-fold the water concentrations of parathion (Miller et al. 1966).

In the terrestrial-aquatic model ecosystem only fish contained parathion (0.1008 ppm) at the end of the 38 day experiment and none of the ecosystem organisms contained paraoxon. Most of the organisms contained radioactivity; 68 percent of the radioactivity in the algae, *Daphnia*, snails, and mosquito larvae was unextractable; in the fish, only 19.7 percent was unextractable, although only 31.5 percent of the unextractable material (6.21 percent of the total piscine radioactivity) was parathion (Yu and Sanborn 1975). Parathion, paraoxon, and *p*-nitrophenol were found in the water at 0.3, 0.47, and 1.4 ppb, respectively. Parathion was estimated to have a half-life of 15 to 16 days in water, and no evidence was found for bioaccumulation in any of the organisms (Yu and Sanborn 1975, Metcalf and Sanborn 1975).

Volatilization-

On petri dishes, parathion in ethanol solution at 35°C was 62 percent volatilized in 90 days (Alessandri and Amormino 1954) while 94 percent of parathion sprayed on peach leaves was lost within one week (Brunson and Koblitsky 1952). Mistric and Gaines (1953) found that wind, sun, and rain, and/or high temperatures significantly increased the volatilization of methyl parathion and parathion, which, if protected from these elements, retained their full insecticidal power for more than 24 hours, and for more than 48 hours, respectively. After one pound per acre of methyl parathion or parathion was sprayed on cotton in Arizona, immediate residues were 106 ppm of methyl parathion and 117 ppm of parathion; after 96 hours, residues had fallen to 3.9 and 5.5 ppm, respectively (Ware et al. 1972).

Polisu and Floru (1967) observed maximum volatilization of parathion at a relative humidity of 40 percent and a temperature between 20 and 50°C; the precise emulsifiers present did not seem to affect the rate of volatilization. Quinby and co-workers (1958) estimated a half-life of less than 0.5 hours for methyl parathion on cotton in Mississippi, where these conditions for maximum volatilization probably hold. Parathion was also lost more rapidly at high temperatures (Mistric and Martin 1956). Encapsulated parathion persisted for up to 70 days on peach foliage, however (Winterlin et al. 1975).

Although the studies cited did not distinguish between volatilization and photolysis, the importance of volatilization or of dust-borne transport is emphasized by the presence of organophosphate pesticides in the atmosphere above the southeastern United States (Stanley et al. 1971).

Persistence:

Data on the persistence of parathion, methyl parathion and parathion metabolites are summarized in Tables 61 and 62 for those studies in which the percent of residue remaining after a specified time could be ascertained. At ordinary levels of application, either to soil or to foliage, both compounds are degraded within weeks if microbial degradation can occur (Lichtenstein et al. 1968), and accumulation even of repeated doses is unlikely (Goldsworthy and Foster 1950). Under conditions of excessive application or incorporation to the soil, however, the ordinary modes of degradation do not appear to function. Simulated spills of concentrated parathion resulted in 15 percent residues after five years (Wolfe et al. 1973) and 0.1 percent after 16 years (Stewart et al. 1971). The residue levels of 13,000 ppm reported by Wolfe et al. (1973) were considered high enough to be lethal and were considered a plausible contamination arising from normal methods of transferring parathion. Microorganisms in the area of the simulated spill did not show adaptation in degrading parathion or enhanced re-

Table 61. PERSISTENCE OF PARATHION IN SOILS

% residue	# of days	% mean loss/day	Conditions	Reference
0.1	5,840	0.02	11.4 lbs/A soil incorporated	Stewart et al. 1971
15	1,825	0.05	30,000 - 95,090 ppm	Wolfe et al. 1973
15	240	0.35	loam	Iwata et al. 1973
7.5	240	0.39	silt loam, low moisture	Ibid.
0	240	0.42	pasture: soil residues	Mol et al. 1972
2.5	180	0.54	Sta. Lucia loam, low moisture	Iwata et al. 1973
2.4	165	0.59	100 lbs/A, clay loam	Koeting and Woodward 1951
3	90	1.08	field; silt loam	Lichtenstein and Schulz 1964
<80	90	0.22	pasture, soil residues	Mol et al. 1972
0	79	1.27	12 lbs/A, clay loam	Koeting and Woodward 1951
0.12	45-75	>1.33	soil applied	Eastman et al. 1971
0.02	45-75	>1.33	foliar spray: soil residues	Ibid.
2	56	1.75	soil	Sacher et al. 1972
0	41	2.44	90°F, 55% relative humidity	Kalkat et al. 1961

Table 61. Cont'd

% residue	# of days	% mean loss/day	Conditions	Reference
5-10	30	3.00	silt loam, 40% moisture, 30°C	Iwata *et al.* 1973
5-10	30	3.20	clay, 40% moisture, 30°C	Ibid.
5-10	30	3.00	sandy loam, 40% moisture, 30°C	Ibid.
0.01	30	3.33	loamy sand, 40% moisture, 30°C	Ibid.
12	14	6.29	soil	Sacher *et al.* 1972
30	12	5.83	Lab: loam soil	Lichtenstein and Schulz 1964
>0	140	<0.71	1 mg/5 gm. in jars	Carlo *et al.* 1952
23	17	4.53	H_2O-percolated soil + 1 ppm para	Lichtenstein *et al.* 1960
80	17	1.18	1 ppm para + NaN_3	Ibid.
80	17	1.18	1 ppm para + autoclaving	Ibid.
0	16	6.25	2 lbs/A, clay loam	Keating and Woodward 1951

Table 42. PERSISTENCE OF METHYL PARATHION, PARAOXON, AND AMINOPARATHION IN SOILS

Compound	% residue	# of days	% mean loss/day	Conditions	Reference
methyl parathion	32-49	60	0.99	20 ppm, 30°C	Baker and Applegate 1970
	21-40	60	1.16	20 ppm, 30°C, irradiated	Ibid.
	20-30	60	1.25	20 ppm, 50°C	Ibid.
	19-25	60	1.30	20 ppm, 50°C, irradiated	Ibid.
	52-54	50	0.94	5 ppm, 30°C	Ibid.
	38-44	50	1.18	5 ppm, 30°C, irradiated	Ibid.
	35-36	50	1.29	5 ppm, 50°C	Ibid.
	18-22	50	1.60	5 ppm, 50°C, irradiated	Ibid.
	3	30	3.23	field: silt loam	Lichtenstein and Schulz 1964
	5	12	7.92	lab: loam soil	Ibid.
	50	11	4.55	ultra-low volume spray	Saini *et al.* 1970
	50	6	8.33	emulsifiable concentrate spray	Ibid.
aminoparathion	0	2	–	lab: loam soil	Lichtenstein and Schulz 1964
paraoxon	0	0.5	–	lab: loam soil	Ibid.

sistance to parathion and the number of bacterial colonies in the vicinity was reduced. A similar observation was made by Kasting and Woodward (1951) who observed that 2 lb/A (2.24 kg/ha) of soil-applied parathion disappeared within 16 days, whereas 100 lbs/A (112 kg/ha) persisted for over 165 days.

Methyl parathion was also said to be retained longer in the soil if the initial concentration was substantial (Naishtein et al. 1973); the authors did not consider soil microorganisms to be the primary means of degradation. Stobwasser (1963) observed that the second year's application of parathion produced lower residues in carrots, even though higher levels were applied, than did the first year's treatment.

Persistence of parathion and methyl parathion were found to depend on the type of soil and the rate of application (Burkhardt and Fairchild 1967). In the laboratory, at 30°C and 40 percent soil moisture, three ppm of parathion remained in Vina loam eight months after 20 ppm had been incorporated. Under the same conditions, Mocho silt loam, Linne clay, and Madera sandy loam retained one to two ppm after 30 days, and Laveen loamy sand retained only 0.2 ppm after 30 days. Changing the moisture resulted in persistence of up to 1.5 ppm in silt loam after eight months instead of after one month. Both microbial and hydrolytic degradation were considered to contribute to the dissipation of the parathion (Iwata et al. 1973).

Harris and Mazurek (1966) compared the activity of pesticides in dry mineral soil, moist mineral soil and muck. Both parathion and methyl parathion were most effective in moist mineral soil and least effective in dry sand or clay, but more effective in moist muck (Harris 1966). When clay was added to sandy loam, the effectiveness of parathion decreased (Harris 1967). Liang and Lichtenstein (1974) compared the toxicity of parathion to fruit flies on sand and on silt loam and observed greater toxicity on sand; parathion toxicity synergized with atrazine if the latter was present at levels of at least 10 ppm. In sandy loam, parathion lost its effectiveness within four weeks (Harris 1969). The addition of detergents increased the two month persistence level of parathion thirteen-fold and the detergents also synergized the toxicity of the pesticide against fruit flies (Lichtenstein 1966).

Leenheer and Ahlrichs (1971) analyzed the kinetics of parathion adsorption on soil organic matter and determined that the initial rate of adsorption was limited by the diffusion of the pesticide onto the adsorptive sites on the outer surfaces of the adsorbent; after about ten minutes, the limiting factor became the diffusion of parathion into the interior of the adsorbent. The fast, reversible adsorption, with low heats of adsorption and high adsorptive capacity on hydrophobic surfaces which was characteristic of parathion was considered indicative of physical adsorption, with Van der Waals bonds between parathion and the adsorbent surfaces (Leenheer and Ahlrichs 1971). Yaron and Saltzman (1972) suggested that parathion cannot displace the strongly adsorbed water molecules in partially hydrated systems, so adsorption occurs mainly on water-free surfaces. In consequence, parathion toxicity in clay or organic soil increases, and its persistence decreases, as increasing soil moisture prevents its adsorption (Naumann 1959, Harris 1964a, Harris 1964b, Lichtenstein and Schulz 1964, Harris and Mazurek 1966, Harris 1967, Chopra and Khullar 1971). Increasing amounts of clay or organic matter also provided more hydrophobic sites on which parathion could be adsorbed (Chopra et al. 1970, Saltzman et al. 1972).

Parathion was more readily adsorbed on organic than on mineral surfaces from aqueous solution, and bonding to organic matter was stronger. Desorption from mineral surfaces was rapid and essentially complete, but only small amounts of parathion were released from organic matter (Saltzman et al. 1972, Kliger and Yaron 1975). Not only did organic matter adsorb more parathion than did clays, but sodium montmorillonite was a better adsorbent than was kaolinite (Saltzman and Yaron 1972).

Addition of organic matter was also found to alter the route of parathion degradation from hydrolysis to nitroreduction, with aminoparathion as the product (Sethunathan 1973).

The persistence of parathion was said to be increased by increasing the pH (Carlo *et al.* 1952) or decreasing the temperature (Chopra and Khullar 1971, Yaron and Saltzman 1972, Baker and Applegate 1970). Methyl parathion added to sandy-clayey soil at 20 mg/kg decomposed in seven days at 20 to 25°C when the soil moisture was 6.5 to 10 percent, but in ten to eleven days when the soil moisture was reduced to 1.7 to 3.4 percent (Obuchowska 1967). Amendation of soil with different ions altered adsorption and the relative magnitude of adsorption in the presence of the ions was: $H^+ > Ca^{++} > Mg^{++} > Na^+$ (Chopra *et al.* 1970).

The persistence of methyl parathion was twice as great in ultra-low-volume sprays as in emulsifiable concentrations when sprayed on foliage in normal agricultural usage (Saini *et al.* 1970). In dusts, increasing the mean particle size of the carrier from 11.8 microns to 23.2 microns speeded degradation by about five percent (Takehara *et al.* 1967a). Methyl parathion was more rapidly degraded from a Zeeklite formulation than from clay L; the rate of degradation varied directly with the total iron content, the amorphous mineral content, and the base exchange capacity of the formulation (Takehara *et al.* 1967b).

Effects on Non-Target Species:

Microorganisms--The effect of parathion and methyl parathion on soil microorganisms are summarized in Tables 63 and 64 and Table 65, respectively. The few data on analogs and metabolites are also included in Table 65.

Capriotti and Martini (1959) found parathion to inhibit the growth of microflora at normal levels of application, but Somer (1970) observed no such inhibition. Cowley and Lichtenstein (1970) observed fungal

Table 63. EFFECTS OF PARATHION ON SOIL MICROORGANISMS

Parameter	Effect	Level		Reference
		Original	PPM	
Bacteria (total)	+	1,000 ppm	1,000	Naumann 1958
″	+	10,000 ppm	10,000	Naumann 1959
″	+	≤50 kg/ha	≤25	Scheffler *et al.* 1952
Nitrifying bacteria	–	ag levels		Capriotto and Martini 1959
″ ″	0	10 kg/ha	~4.5	Pechon *et al.* 1951
Nitrification	0	250 ppm	250	Wilson 1954
″	–	1,500 ppm	1,500	Ibid.
″	0	1,500 ppm	1,500	Ibid.
Nitrifying bacteria	+	50,000 ppm	50,000	Naumann 1959
Nitrogen fixing bacteria	+	50,000 ppm	50,000	Ibid.
Aerobic cellulolytic bacteria	0	10 kg/ha	~4.5	Pechon *et al.* 1951
Cellulolytic	+	50,000 ppm	50,000	Naumann 1959
Anaerobic	+	50,000 ppm	50,000	Ibid.
Fungi	–	.0042	40	Scheffler *et al.* 1952

+ = stimulation
0 = no effect
– = inhibition

Table 64. EFFECTS OF PARATHION ON SPECIES OF MICROORGANISMS

Parameter	Effect	Level Original	 PPM	Reference
Actinomycetes	0	2,688 kg/ha	<1.3	Hubbell et al. 1973
Aspergillus fumigatus	–	<2 ug/ml	<2	Cowley and Lichtenstein 1970
Aspergillus respiration	–	10^{-3} M		Eder 1963
Saccharomyces ferment	+	10^{-3} M		Ibid.
Pseudomonas [illegible]	–	10,000 ppm	10,000	Yasue 1956
Pseudomonas [illegible]	0	1,000 ppm	1,000	Ibid.
Fusarium oxysporum	–	<10 ug/ml	<10	Cowley and Lichtenstein 1970
Escherichia coli	0	≤10,000 ppm	≤10,000	Yasue 1956
Azotobacter	0	10 kg/ha	<5	Pochon et al. 1951
Azotobacter	0	4–400 ppm	4–400	Gill et al. 1970
Nitrobacter agilis	–	10 ug/ml	10	Garretson and San Clemente 1968
Infusoria	–	.025–.052	250–500	Popov 1958

+ = stimulation
0 = no effect
– = inhibition

Table 61. EFFECTS OF p-NITROPHENOL AND METHYL PARATHION ON SOIL MICROORGANISMS

Parameter	Effect	Level Original	 PPM	Duration	Reference
p-nitrophenol:					
Soil microorganisms	+	160 ppm pure	160		Naumann 1970d
Actinomycetes	−	.22% formulated	2200		Ibid.
Nitrifying bacteria	−	.39% formulated	3900		Ibid.
Cellulolytic bacteria	−				Ibid.
Infusoria	−	.025-.05%	250-500	10 minutes	Popov 1958
Methyl parathion:					
Soil microorganisms	+	300 ppm	300		Naumann 1970
Bacteria	+	0.5-200 x ag level		≥26 weeks	Naumann 1970a
Bacteria	0	5 ppm	5		Naumann 1970b
Bacteria	−	0.5% formulated	5000		Naumann 1970d
Bacteria, gram neg.	−	75-250 kg/ha	34-112	<2 weeks	Naumann 1971
Bacteria, gram neg.	+	75-250 kg/ha	34-112	2-3 weeks	Ibid.
Bacteria, gram neg.	−	75-250 kg/ha	34-112	>3 weeks	Ibid.
Autochthones	+	75-250 kg/ha	34-112	5 weeks	Ibid.
Actinomycetes	+	.5-200 x ag level		<26 weeks	Naumann 1970a
Azotobacter	−	4-400 ppm	4-400		Gil et al. 1970c
Soil respiration	−	≤500 ppm	≤500	several hours	Naumann 1970c
Soil respiration	+	≥500 ppm	≥500	several hours	Ibid.

+ = stimulation 0 = no effect − = inhibition

inhibition at levels approximating field applications of parathion and of the seventeen fungi tested, none was able to use parathion as a source of carbon or phosphorus. Since the data were obtained under laboratory conditions, the authors warned against extrapolation to field conditions. Parathion was tested for mutagenicity in *Escherichia coli* K-12 and was not mutagenic (Mohn 1973), but did inhibit aflatoxin synthesis by *Aspergillus* (Hsieh 1973).

The effects of methyl parathion on soil microorganisms have been thoroughly investigated by Naumann (1970a, 1970b, 1970c, 1970d, 1971), as summarized in Table 65. Naumann noted that nitrogen-fixing bacteria were most affected by methyl parathion (formulated as Wofatox), whereas denitrifying bacteria and soil algae tended to remain unaffected. Bacterial populations tended to increase and fungal populations to decrease (1970b). When soil respiration was measured in a Warburg apparatus, 500 ppm or less of methyl parathion resulted in a decrease in soil respiration, followed several hours later by an increase; three separate additions of 5,000 ppm ended the respiratory stimulation after 12 weeks. The effects were similar in clay, sand, or compost, but were least marked in clay. Formulated methyl parathion, in contrast to the pure compound, depressed actinomycetes, nitrogen-fixing bacteria, cellulolytic bacteria, denitrifying bacteria, and soil algae (1970d). The data make it very clear that methyl parathion served as a substrate for soil microorganisms (1970c). Mohn (1973) considered methyl parathion a probable mutagen after testing it in *E. coli*.

Despite the mélange of techniques, it would appear that parathion and methyl parathion stimulated the soil microflora, particularly bacteria. The source of such stimulation is those organisms which, using parathion or methyl parathion as energy sources, multiply in their presence. Since only a small proportion of the total soil microflora, and even of the species known to degrade these pesticides, could be expected to have the actual capability of using parathion as an energy source, the inhibition of pure cultures by parathion is not inconsistent with overall stimulation of soil respiration.

When algae (*Anacystis nidulans*, *Scenedesmus obliquus*) and protozoa (*Euglena gracilis*, *Paramecium bursaria*, *Paramecium multimicronucleatum*) were exposed to parathion at one ppm for seven days, neither adverse effects nor any metabolites were found, although parathion was concentrated from 50 to 116-fold (Gregory et al. 1969). Cabejszek and Maleszewska (1970) found that 0.16 to 0.80 mg/liter (0.16 to 0.80 ppm) of methyl parathion decreased oxygen consumption by aquatic organisms within 30 minutes, but Luczak and Maleszewska (1966) observed no inhibitory effect from methyl parathion formulated as Wofatox at ten mg/liter (10 ppm), and observed stimulation of nitrate-reducing bacteria at 250 mg/liter (250 ppm).

Plants--Parathion and methyl parathion are not considered to be phytotoxic (Martin 1968). At two lbs/A (2.24 kg/ha), however, parathion decreased the stands of cucumbers, bush beans, and turnips, and reduced the quality of peas, cauliflower, and Swiss chard (Stitt and Evanson 1949). Sprays were less damaging than root drenches (Dennis and Edwards 1963). In quartz sand at 30 ppm (equivalent to 16.8 kg/ha) parathion increased the respiration of corn root tips (Lichtenstein et al. 1962). Leaf damage to sorghum was reported from both parathion and methyl parathion (Meisch et al. 1970). Parathion did not, however, accumulate in carrots under conditions producing residues of diazinon and other organophosphates (Bro-Rasmussen et al. 1969). Anaphase inhibition was caused by 0.0001 to 0.05 percent solutions of parathion in the onion, *Allium cepa*; c-mitoses, chromosome and chromatid breaks, micronuclei and anaphase bridges were also observed (Ravindran 1971).

Invertebrates--Methyl parathion at ten ppt killed 80 percent of Louisiana red crayfish (*Procambarus clarkii*) within five days in the laboratory, but 100 ppb had no effect in the field (Hendrick et al. 1966).

Aquatic invertebrates were killed by levels of parathion ranging from 0.004 ppm in the caddis fly (*Arctopsyche grandis*) to 0.032 ppm in the stone fly (*Pteronarcys californica*); only endrin was consistently more toxic (Gaufin et al. 1961, 1965). Luedemann and Neumann (1962) pointed out that, although the organophosphates were less toxic to fish than the organochlorines, both classes were equally toxic to piscine food organisms.

The effects of organophosphates on soil invertebrates have been treated in the excellent review of Edwards (1973) who concluded that, although not as toxic to mites as many organochlorines, organophosphates are particularly toxic to predatory mites. Effects on other soil invertebrates were not well defined. Parathion has long been considered highly dangerous to bees (Eckert 1948), but was acutely less toxic to honeybees in the laboratory than dieldrin, azinphosmethyl, carbaryl, phorate, or DDT (Johansen 1961). Dusts were twice as toxic to bees as sprays (Johansen et al. 1957).

Fish and amphibians--The toxicity of parathion to fish varies from a 96 hour TL_m (median tolerance limit) of 0.055 ppm in guppies (*Lebistes reticulatus*) to 3.7 ppm in goldfish (*Carassius auratus*), with bluegills (*Lepomis macrochirus*) and fatheads (*Pimephales promelas*) intermediate (Pickering et al. 1962). Leudemann and Neumann (1961) found three mg/liter (3 ppm) of parathion to be lethal to all rainbow trout (*Salmo irideus*) and pike fry (*Esox lucius*) which were exposed while the no-effect levels were 0.8 mg/liter (0.8 ppm) for trout and 1.0 mg/liter (one ppm) for pike. Spermatogenesis in guppies was decreased by exposure to 0.1 to one ppm of parathion for more than six days; ten ppm were lethal within hours (Billard et al. 1970).

Eisler (1970b) found that methyl parathion was less toxic to several species of fish than were the organochlorines, Phosdrin, malathion, or DDVP. The 96 hour toxicity level of parathion was 5,200 to 75,800 ppb in piscine species including the American eel (*Anguilla rostrata*), blueheads (*Thalassoma bifasciatum*), striped killifish (*Fundulus majalis*), and striped mullet (*Mugil cephalus*). Mulla and co-workers (1963) found methyl parathion, at levels used in controlling mosquito larvae, to be nontoxic to mosquitofish. Parathion applied to ponds at 0.1 lb/A (0.11 kg/ha) killed 22 percent of the mosquitofish (*Gambusia affinis*) added 30 hours after treatment (Mulla and Isaak 1961). The toxicity was greater over a period of 240 than over 96 hours in the estuarine mummichog, *Fundulus heteroclitus* (Eisler 1970a). Toxicity was increased by increasing water temperature from 10° to 30°C, increasing salinity, or decreasing the pH. While the latter alterations in toxicity might be due to nonspecific stress effects, the increased toxicity after 45 minutes was undoubtedly due to paraoxon formation; greater toxicity of longer time periods suggests cumulative action either due to accumulation of toxicants, as with organochlorines, or progressive toxicity, as in the neurotoxicity evoked by some organophosphate compounds.

Lee and Russell (1969) induced respiratory changes in goldfish with 0.56 mg/liter (0.56 ppm) of methyl parathion although it took 3.20 mg/liter (3.2 ppm) for 65 days to kill the fish. Burke and Ferguson (1969) found that parathion was less toxic in flowing than static solutions, presumably because of the accumulation of paraoxon under the latter conditions. DDT, toxaphene and endrin, which do not require activation, were more toxic in flowing solutions. Mount and Boyle (1969) hypothesized that more resistant species of fish might well accumulate parathion to some extent, although the catfish they studied (*Ictalurus nebulosus*) did not.

Parathion caused nerve cell injury in both carp (*Cyprinus carpio* L.) and river crabs (*Cambarus affinis* Say) in a study by Kayser et al. (1962). No data on piscine carcinogenesis, mutagenesis, or teratogenesis were found. Resistance to chlorinated hydrocarbon insecticides did not confer parathion resistance on sunfish or shiners (Minchew and Ferguson 1969).

Amphibians were found to be relatively resistant to organophosphates (Mulla 1962). In the toad, *Bufo viridis*, a lethal injection into the dorsal lymph sac required 967 mg/kg of parathion or [illegible] mg/kg of paraoxon (Edery and Schatzberg-Porath 1960).

Birds--The acute oral LD_{50} of methyl parathion is 6.12 to 16.3 mg/kg in mallards and 5.7 to 11.9 mg/kg in pheasants (Tucker and Crabtree 1970). Parathion has an acute oral LD_{50} of 0.125 to 0.250 mg/kg in fulvous tree ducks, 1.37 to 2.96 mg/kg in mallards, and 12.4 mg/kg in pheasants (Tucker and Crabtree 1970). Keith and Mulla (1966) found no definite toxicity when mallards were fed five ppm parathion daily for five to nine weeks, but 25 ppm were essentially lethal. Heath and co-workers (1972) calculated the LC_{50} for five days of treated feed followed by three days of clean feed to be 90 ppm of methyl parathion in bobwhites, 682 ppm for mallards, 46 ppm for Japanese quail, and 116 ppm for pheasants. For parathion, the corresponding LC_{50} was 44 ppm in Japanese quail, 364 ppm in pheasants, with bobwhites and mallards intermediate. Methyl mercury (Moradren) was found to potentiate the action of parathion in coturnix quail (Dieter and Ludke 1975).

Parathion was teratogenic in chickens if injected into the eggs (Upshall *et al*. 1968, Roger *et al*. 1969, Yamada 1972, Reis *et al*. 1971, Dunachie and Fletcher 1969, Meiniel 1974) and in Japanese quail when eggs were dipped into a ten percent solution (Meiniel *et al*. 1970, Meiniel 1973). Sterilization and feminization of treated offspring were also observed (Lutz-Ostertag *et al*. 1970).

Mammals--Methyl paraoxon and paraoxon inhibit acetylcholinesterase in mammals, and poisoning is due to the resulting potentiation of neural synaptic transmission by acetylcholine. Since the active agent in cholinesterase inhibition is not the phosphorothionate itself, species differences in sensitivity to parathion and methyl parathion may be due to differing rates of conversion to the analogs (paraoxon and methyl paraoxon), differing sensitivities of the cholinesterases to inhibition, or differing rates of metabolism of the oxygen analog (Murphy *et al*. 1968). A succinct review of the mechanisms of action and toxicity of the organophosphates is presented by Fest and Schmidt (1973).

Both parathion and methyl parathion are readily absorbed through the lungs or skin as well as by ingestion. The minimum toxic level for a human adult is estimated at 7.5 mg per day for parathion and somewhat over 19 mg per day for methyl parathion (Rider *et al*. 1969). Faerman *et al*. (1969) found hematological abnormalities in workers producing organophosphates: iron-deficiency anemia, lower hemoglobin content, and erythrocyte degeneration were observed. The acute toxicity of parathion in man is sufficiently great to warrant the conclusion that, regardless of the expected degree of occupational exposure, almost any person who works with parathion is in danger of being poisoned if he is careless (Arterberry *et al*. 1961).

The acute oral LD_{50} of parathion is 13 mg/kg in male and 3.6 mg/kg in female rats; that of methyl parathion, 14 mg/kg in male and 24 mg/kg in female rats (Martin 1968, Gaines 1969). The acute oral LD_{50} of parathion was 11 mg/kg in mice (Klimmer and Pfaff 1955), 22-24 mg/kg in mule deer and 28 to 56 mg/kg in domestic goats (Tucker and Crabtree 1970). The acute dermal LD_{50} of parathion in rats is 21 mg/kg in males and 6.8 mg/kg in females (Martin 1968).

In tissue culture of normal cells (human skin fibroblasts) and malignant cells (HeLa), it was observed that the direct cellular toxicity of parathion was greater than that of paraoxon. The direct cellular toxicity of *p*-nitrophenol, the major terminal residue of parathion, was as great as that of parathion itself (Litterst and Lichtenstein 1971).

Aldrin, chlordane, and DDT were found to protect somewhat against organophosphate poisoning (Triolo and Coon 1966, Williams *et al*. 1967, Chapman and Leibman 1971). Triolo, Mata and Coon (1970) hypothesized that rat serum aliesterases bind paraoxon, and thus the well established enhancement of serum esterases by organochlorine insecticides mediated paraoxon detoxification. Parathion inhibits the metabolism of benzo(a)pyrene in rats (Weber et al. 1974).

Parathion is embryotoxic in all species studied. When female rats were given methyl parathion three days before parturition, methyl paraoxon was found in all fetal tissues within twenty minutes (Ackermann 1974, Ackermann and Engst 1970). Rat fetuses treated to day 17 were, however, found to have normal levels of acetylcholinesterases, suggesting that younger fetuses cannot convert parathion to paraoxon (Talens and Wooley 1973). Tanimura et al. (1967) reported normal litters from rats treated on day 12 of pregnancy. Leibovich (1973) reported decreased numbers of neonates and decreased survival among rats born to dams who were fed parathion (0.1 to ten mg/kg) throughout pregnancy. Embryotoxicity, but not teratogenesis, was reported in offspring of female rats fed 1.5 mg/kg/day of parathion for one week following mating (Noda et al. 1972). Implantations decreased and resorptions increased. Kimbrough and Gaines (1968) found parathion to be teratogenic as well as embryotoxic in rats only if maternal toxicity occurred. In mice, methyl parathion induced cleft palate (Tanimura et al. 1967). Rats were reportedly more sensitive to parathion treatment in early pregnancy than were hamsters (Lauro et al. 1969). Data on other species of laboratory mammals were not available.

The sum of these data is that parathion is acutely toxic and exerts considerable stress on the maternal organism; direct effects on the embryo have not been ruled out, but have not been seen in the rat. There are no data available suggesting that parathion and methyl parathion are either carcinogenic or mutagenic. Data to the contrary were also not available.

Conclusions:

The major hazard of parathion and methyl parathion in ordinary usage is clearly in their acute toxicity to mammals, birds, beneficial arthropods and fish. When applied at reasonable levels in the natural environment, parathion and methyl parathion are degraded within weeks. In the interim, transport can occur from soil into water and from water into sediment. Volatilization inevitably occurs from soil surfaces and from foliage, and quantifiable residues are taken up by food plants. Nevertheless, the relatively rapid detoxification of these compounds by microbial degradation and by hydrolysis assures dissipation.

Under conditions of bulk disposal, however, degradation is significantly retarded (Halshtein et al. 1973) even when the massive treatments are soil-mixed, as would occur in spills. If 31 pounds per acre are applied, residues are found for sixteen years (Stewart et al. 1971), while potentially lethal residues are found in the top inch of soil for five years after the equivalent of 190,000 lbs/A were applied. These data argue strongly against bulk disposal of parathion in the soil. Moreover, there are too few data available on the persistence, soil toxicity, and motility of *p*-nitrophenol and other metabolites of parathion to permit evaluation of their effects on the environment.

Nonbiological degradation of parathion production wastes is feasible, but requires extensive cleanup procedures (Stutz 1966). More plausible would be controlled microbial degradation, preferably in a two-phase process which permits anaerobic detoxification followed by aerobic ring-cleavage as described by Sethunathan (1973a, 1973b) and by Sethunathan and Yoshida (1973a, 1973b). No data are available on the feasibility of such a process on a large scale.

Malathion

Malathion is the common name for *S*-1,2-bis-(ethoxycarbonyl)ethyl *O*,*O*-dimethyl phosphorodithioate, introduced by the American Cyanamid Company in 1950 as Malaphos. Its common name in West Germany and South Africa is mercaptothion; in the USSR, carbofos. Malathion is prepared by the addition of *O*,*O*-dimethyl phosphorodithioic acid to diethyl maleate in the presence of hydroquinone (Martin 1968). It is yellow to dark brown liquid with a melting point of 2.85°C, a boiling point of 156° to 157°C at 0.7 mm mercury, and a vapor pressure of 4×10^{-5} mm mercury at 30°C. Its solubility in water is 145 ppm at room temperature and light petroleum oils are soluble to about 35 percent malathion. It is formulated as emulsifiable concentrates, dusts, and atomizing concentrates for use as a nonsystemic insecticide and acaricide.

Degradation:

Walker and Stojanovic (1973) analyzed the relative contributions of chemical and biological reactions to the degradation of malathion in soil. They concluded that microbial degradation predominated, but that increasing pH or soil organic matter increased the importance of hydrolysis. The relative contributions of chemical and biological reactions in three soils are shown in Table 66, and data on biological degradation are summarized in Table 67.

Biological--Neither *Trichoderma viride* nor *Pseudomonas* converted malathion to malaoxon, although both degraded malathion (Matsumura and Boush 1966). Soil fungi (*Penicillium notatum*, *Aspergillus niger*) were able to degrade three mg malathion per 100 ml nutrient solution (30 ppm), but *Rhizoctonia solani* was inactivated at this level, although active at two mg malathion per 100 ml nutrient solution (20 ppm) (Mostafa et al. 1972b). *Rhizobium trifolii* degraded 91.5 percent of mala-

Table 56. DEGRADATION OF MALATHION IN THREE SOILS OVER A 10 DAY PERIOD (WALKER AND STOJANOVIC 1973).

	Total Degradation (%)	% Chemical	% Biological
Loam	100	9	91
Sand	59	5	95
Clay	100	23	77
Loam-water	85	60	40
Sand-water	29	3	97
Clay-water	71	47	53

Table 67. BIOLOGICAL DEGRADATION OF MALATHION

Organism	Products	Reference
Rhizobium trifolii " leguminosarum	thiophosphates, 5 other metabolites	Mostafa et al. 1972a
Penicillium notatum Aspergillus niger Rhizoctonia solani Rhizobium trifolii Rhizobium leguminosarum	thiophosphate, monomethyl phosphate, dimethyl phosphate, dimethyl phosphorothioate, dimethyl phosphorodithioate, malathion diacid	Mostafa et al. 1972b
Aspergillus oryzae	β-malathion monoacid; ethanol	Lewis et al. 1975
Soil	thiomalic acid, dimethylthiophosphoric acid, diethylthiomalate.	Konrad et al. 1969
Soil esterase	malathion monoacid	Getzin and Rosefield 1968
Heterogeneous aquatic bacteria	β-malathion monoacid, dicarboxylic acid O,O-dimethyl phosphorodithioic acid, diethyl maleate	Paris et al. 1975
Pseudomonas sp. Trichoderma viride	no malaoxon	Matsumura and Boush 1966
Arthrobacter	carboxylic half-ester, dicarboxylic acid, potassium dimethyl phosphorothioate, potassium dimethyl diphosphorothioate, unidentified products	Walker and Stojanovic 1974
Cow	dimethyl phosphate	O'Brien et al. 1961

thion within one week with only 0.5 percent being converted to malaoxon (Mostafa et al. 1972a). Getzin and Rosefield (1968) reported a stable, cell-free soil enzyme which converted malathion to its monoacid; the enzyme was resistant to heat, dessication, and microbial destruction. Sethunathan and Yoshida (1973a) identified a *Flavobacterium* which degraded parathion and diazinon but not malathion. In alkaline soils, adsorption reportedly preceded degradation (Konrad et al. 1969).

Malathion was readily degraded by sewage lagoon microorganisms (Halvorson et al. 1971). In activated-sludge sewage systems, the volume of organic matter was better correlated with the degree of degradation than was the volume of water. A ratio of malathion:microorganisms of 1:5 or less inhibited microbial degradation (Randall et al. 1967). In surface water the rate of malathion degradation by natural and sewage microorganisms increased for five days, presumably reflecting changes in microbial populations (Jirik et al. 1971).

Malathion sufficed as the sole source of carbon for a heterogeneous collection of bacteria from aquatic culture, but degradation proceeded only to the malathion-*beta* monoacid, which remained stable for at least 4.5 months (Paris et al. 1975). An aquatic fungus, *Aspergillus oryzae*, also converted malathion to its monoacid, producing ethanol as a by-product, which served as a carbon source for the fungi. Fungal transformation was, however, much slower than the bacterial transformation (Lewis et al. 1975). In a microbial salt-marsh ecosystem, malathion was rapidly decomposed by indigenous bacteria, with a relatively insignificant contribution by those bacteria which use malathion as their sole source of carbon. The major agents of degradation were carboxyesterases and some phosphatases (Bourquin 1975).

Paraoxon residues on plants inhibited malathion dissipation from plums, tomatoes, and string beans after harvest; neither parathion nor malathion itself was inhibitory (Koivistoinen et al. 1964). Aldrin pretreatment, in contrast, increased malaoxon degradation (Cohen and Murphy 1974). Wheat seed-coat enzymes converted malathion to its thiolate and phosphate (Rowlands 1966), and in rice grains malathion is converted to thiophosphoric acid (Tomizawa *et al*. 1962).

Malaoxon is produced from malathion by mouse liver (O'Brien 1957), where its inactivation depends on an esterase highly sensitive to other organophosphates. Therefore, the toxicity of malathion can be sharply increased by the presence of other organophosphates (Cook *et al*. 1957, 1958). A lactating cow which was fed 63 ppm malathion for three days excreted 77 percent within one week; the remainder had not been excreted after three weeks (O'Brien *et al*. 1961).

In an aquatic-terrestrial ecosystem, malathion was exceptionally degradable: no traces of the parent compound were found in any of the organisms. The fish, snails, and mosquito contained several uncharacterized metabolites, which were also found in water (Metcalf and Sanborn 1975).

Photolytic--Mosher and Kadoum (1972) analyzed the dissipation of malathion from surfaces under the influence of light. Themost rapid degradation was observed with infra-red light and was due to the heat generated (119°C). Far ultraviolet was a more effective light source for degrading malathion, than near ultraviolet light. Decomposition products included malaoxon, malathion monoacid, malathion diacid, O,O-dimethyl phosphorothioic acid, dimethyl phosphate, phosphoric acid, and unidentified products. Malathion disappeared most rapidly from glass beads, and least rapidly from wheat grains. Sorghum grains were intermediate (Mosher and Kadoum 1972).

Anthraquinone accelerated the photodecomposition of malathion on silica gel chromatographic plates (Ivie and Casida 1971). After 16 hours of exposure to ultraviolet light at 37°C, 66 percent of the malathion had disappeared from ladino clover (Archer 1971). Wolfe and co-workers (1975), using wave lengths above 290 nm, estimated the photolytic half-life of malathion to be 990 hours (41.25 days) in water at pH 6, but only 16 hours in Suwannee river water which contained large amounts of colored material.

Chemical--Malathion was more rapidly degraded in clay loam in which heat-labile substances had been destroyed than in the original soil (Getzin and Rosefield 1968). In water at pH 10, malathion decomposed to the dimethyl phosphate and succinic or thiosuccinic esters (Shevchenko *et al.* 1974). Degradation is extremely rapid at high pH (Paris and Lewis 1973) but proceeds quite slowly at low pH, and Wolfe *et al.* (1975) reported that malathion persisted for up to two weeks in oxygen-saturated acidic water. Spiller (1961) could not detect degradation after 12 days at pH 5.0 to 7.0, and similar results were obtained for pH 2.0, 4.0, and 6.0 (Konrad et al. 1969). In neutral solution, malathion was 70 percent hydrolyzed in one week (Cowart *et al.* 1971).

Of malathion formulated in Zeeklite (a quartz cristobalite) 8.4 to 9.2 percent had decomposed after 30 days at 40°C, while 36.9 percent of the malathion formulated on clay L had decomposed in the same period (Takahara *et al.* 1967b). The decomposition of dusts was not affected by moisture but was catalyzed by copper, lead, mercury or tin (in decreasing order of efficacy) while the decomposition of emulsions increased with increasing moisture (Yamauchi *et al.* 1959). When malathion was applied to a calcareous (alkaline) West Point loam at the equivalent of 11,227 kg/ha (five tons per acre), carbon dioxide evolution decreased if analytical grade malathion was used, but increased when formulated malathion was used, presumably due to degradation of the carrier (Stojanovic *et al.* 1972a).

Malathion was completely degraded by treatment with liquid ammonia and metallic sodium or lithium (Kennedy *et al.* 1972a). Treatment with 8*N* sodium hydroxide decomposed malathion to inorganic phosphate, while 15*N* ammonium hydroxide partially decomposed it (Kennedy *et al.* 1972b). Ozone treatment of malathion-contaminated water reportedly decreased the toxicity of the water (Gabovich and Kurennoi 1966).

Physical--Cobalt-60 *gamma*-irradiation of 0.1 to 5.0 megarads decomposed 71 percent of malathion in hexane (Lippold *et al.* 1969). The heat produced by infrared light also decomposed malathion (Mosher and Kadoum 1972). Stojanovic *et al.* (1972b) identified the degradation products of malathion heated to 300°C to be the diethyl esters of succinic, maleic, and/or fumaric acids. Pure malathion was 25 percent decomposed by heating to 663°C, but formulated malathion was 25 percent decomposed only at 715°C (Kennedy *et al.* 1969). At 900°C, malathion decomposed to form carbon monoxide, carbon dioxide, sulfur dioxide, hydrogen sulfide, and oxygen (Kennedy *et al.* 1972b).

Transport:

Within soil--At relative humidity greater than 40 percent, malathion penetrated into montmorillonite (Bowman *et al.* 1970). Between 4.2 and 5.3, the pH of the clays did not affect adsorption, but calcium and magnesium clays adsorbed less malathion than potassium clays. Malathion adsorption was positively correlated with organic matter content, humic acid content, and cation exchange capacity (MacNamara and Toth 1970). On pond sediments and watershed soils, malathion was more readily adsorbed than either phorate or carbaryl (Meyers *et al.* 1970). No data were available on the movement of malathion through soil, and little significance can be attached to the effects of such movements since malathion is ordinarily degraded rapidly. But the absence of degradation of adsorbed malathion (Bowman *et al.* 1970) suggests that movement of malathion with soil, as on washoff, is possible. No data were available on the transport of malathion metabolites and no data were available on the movement of malathion or its metabolites between soil and water or between water and sediment.

Into organisms--No malathion residues were found in milk after treatment of cows with ultra-low-volume sprays for horn fly control (Oehler et al. 1969), but when malathion was used to disinfect stables, 0.08 to 0.28 ppm were detected in the milk for up to three months (Milhaud et al. 1971).

Carp exposed to 2.5 mg/liter of malathion (2.5 ppm) for four days accumulated 7.91 ± 2.27 mg/kg in their livers (Bender 1969). In a salt marsh in northwestern Florida, 0.42 kg/ha were applied in three foggings at two week intervals and blue crabs (*Callinectus sapidus*), grass shrimp (*Palaemonetes vulgaris, Palaemonetes pugio*), pink shrimp (*Penaeus duorarum*) and sheepshead minnows (*Cyprinodon variegatus*) confined in the treated area contained no measurable residues of malathion, nor did snails (*Littorina irrorata*), but plants (*Juncus* sp.) contained 0.05 to 0.10 ppm malathion for up to 14 days. Water retained traces of malathion for one day (Tagatz et al. 1974).

Volatilization:

Ethanol solutions of malathion were evaporated to dryness on watch glasses and kept at 35°C for 90 days. Not more than 7.5 percent of the malathion volatilized in the first fifteen days; 41 to 49 percent had volatilized after 90 days; and the lower levels of volatility were observed with malathion of greater purity (Alessandrini and Amormino 1954).

Persistence:

The persistence of malathion in soil, in water, and on organisms is summarized in Table 68. Malathion residues were found in 43 percent of the soils sampled in five west Alabama counties (Albright et al. 1974). Malathion was more persistent in muck than in soil with low organic matter content and soil residues of malathion after eight days were said to be three percent (Lichtenstein 1965). Krishnan and coworkers (1958) claimed five week residual effectiveness for water-dispersible malathion powders. Chopra and Girdhar (1971) found persistence to increase with increasing pH, decreasing relative humidity,

Table 68. PERSISTENCE OF MALATHION IN SOIL, IN WATER, AND ON ORGANISMS

% residue	# days	% mean loss/day	Conditions	Reference
0	10	10	loam	Walker and Stojanovic 1973
1	10	10	sand	Ibid.
0	10	10	clay	Ibid.
15	10	8.5	loam dilutions	Ibid.
71	10	2.9	sand dilutions	Ibid.
25	10	7.5	clay dilutions	Ibid.
0	2	50	soil; bioassay	Lange and Schultz 1963
4	21	<5	25 mg/100 ft^2; bioassay	Krishnan et al. 1958
0	3	33	water; field conditions; bioassay	Mulla 1963
0	2	50	water; laboratory; bioassay	Ibid.
25	7	10.4	river water in jars	Eichelberger and Lichtenberg 1971
10	14	6.4	river water in jars	Ibid.
0	28		river water in jars	Ibid.
3	8	12.1		Lichtenstein 1963
50	0.87	57.5	29.4°C, ULV spray on cotton	Saini et al. 1970
50	0.63	79.4	40.6°C, ULV spray on cotton	Ibid.
50	0.41	122.0	29.4°C, EC spray on cotton	Ibid.
50	0.33	151.5	40.6°C, EC spray on cotton	Ibid.
2.7	14	6.9	calves' hides	March et al. 1956
8	28	3.3	neutral aqueous medium	Cowart et al. 1971
0	16.7	5.9	henhouse litter	Galley 1972

increasing concentration, and decreasing exposure to ultraviolet light. On plants, ultralow volume sprays were approximately twice as persistent as emulsified concentrations (Saini et al. 1970).

In water, malathion is hydrolyzed almost instantly at pH 12, but not at all at pH 5 to pH 7 (Spiller 1961). Paris and Lewis (1973) suggested that malathion may be relatively persistent in aquatic systems because it is not rapidly destroyed at neutral pH but in samples of river water ranging from pH 7.3 to 8.0, malathion residues after seven days were 25 percent of the original level and after four weeks no malathion was detectable (Eichelberger and Lichtenberg 1971). Menzie (1972) characterized the persistence of malathion in water as "variable", but noted that it persisted less than one day in fish. In cattle, 2.7 percent of ^{32}P-malathion remained in the hide after two weeks (March et al. 1956).

Very little has been published on the fate of non-insecticidal metabolites of malathion. Lenon and co-workers (1972) reported the presence of an unidentified metabolite of malathion in all of 49 cisterns tested on the Virgin Island while malathion itself was present in only two of the cisterns, at levels of 0.01 ppb and 0.14 ppb. Paris et al. (1975) reported that both β-malathion monoacid and malathion diacid were stable for 4.5 months in aquatic cultures.

Effects on Non-Target Species:

Microorganisms--Malathion was not mutagenic in *Escherichia coli* under conditions in which a number of organophosphates were mutagenic (Mohn 1973). Yurovskaya and Zhulinskaya (1974) claimed that any effects of malathion on soil microorganisms were only temporary.

The effects of malathion on aquatic microorganisms are summarized in Table 69. Algae were considered less sensitive to malathion than either protozoa or rotifers (Hanka-Rybicka and Stanislawska 1977). DaSilva and co-workers (1975) reported that malathion at a level of 100 ppm initially

Table 49. EFFECTS OF MALATHION ON AQUATIC MICROORGANISMS

Parameter	Effect	Level Original	PPM	# days observed	Conditions	Reference
Algal growth:		100 ppm	100	28	culture	Da Silva *et al.* 1974
Anabaena cylindrica	+			28		Ibid.
Aulosira sp.	0			28		Ibid.
Nostoc	–			28		Ibid.
Nostoc muscorum	0			28		Ibid.
Tolypothrix tenuis	+			28		Ibid.
Westiellopsis sp.	+			28		Ibid.
Calothrix elenkinii	+			28		Ibid.
Chlorogloea fritschii	+			28		Ibid.
Cylindrospermum muscicola	–			28		Ibid.
	+			1/24		Ibid.
Euglena gracilis	0					Fernandova and Kremery 1967
	–	high				Moore 1970
Chlorella pyrenoidosa	–	100 ppm	100			Christie 1969
Bacteria	–	>0.1%	1000		sewage lagoons	Steelman *et al.* 1967
Bacteria	+	≥0.1%	1000		water	Lucas *et al.* 1973
Plankton	–	>5 mg/l	>5000		water	Ibid.

+ = stimulation
0 = no effect
– = inhibition

inhibited algal growth and subsequently stimulated it. One species among nine algae tested, (*Cylindrospermum muscicola*) was already exceeding control growth after one hour; one species (*Nostoc* derived from *Collematenax*) never reached the control levels of growth. The other seven species (See Table 69) were below control levels after one hour, and well above after 28 days. In sewage disposal lagoons, levels of malathion as low as one ppm caused 0.83 percent mortality (Steelman et al. 1967).

The effects of malathion on soil processes and soil microorganisms are summarized in Table 70. At 10 ug/ml (10 ppm) malathion completely inhibited *Nitrosomonas europaea*, but 1000 ug/ml (1000 ppm) were required to inhibit *Nitrobacter agilis* (Garretson and San Clemente 1968). In an upland Huvalickes clay loam, malathion decreased the total nitrogen concentration of the soil, and first decreased, but then increased, soil levels of phosphorus and calcium (Vicario 1972). Gram-positive bacteria were more sensitive to malathion than gram-negative bacteria (Nestor 1972) and malathion was toxic both to entomogenous fungi and to pathogenic fungi (Hulea and Piticas 1973). Aflatoxin synthesis in *Aspergillus* was inhibited by malathion (Hsieh 1973).

At five tons per acre (11,227 kg/ha), formulated malathion strongly inhibited bacteria and streptomyces, and slightly inhibited fungal growth. Malathion itself inhibited bacterial growth slightly, and stimulated streptomyces (Stojanovic et al. 1972a).

Invertebrates--Malathion applied to white clover (*Trifolium repens*) at 1.25 lbs/A (1.39 kg/ha) of wettable powder or emulsifiable concentrate was not residually toxic to honeybees (Clinch 1969), but 0.50 to 0.75 lbs/A (0.61 to 0.84 kg/ha) was toxic to all bees in the field (Anderson and Atkins 1958). A 48 hour exposure to malathion reportedly increased tolerance of copepods in the contaminated areas to malathion (Naqvi and Ferguson 1968, 1969). In treated and control areas of a pine hardwood forest, microarthropod populations did not differ one

Table 70. EFFECTS OF MALATHION ON SOIL ORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	Level PPM	# days observed	Conditions	Reference
Nitrification	–	1000 µg/ml	1000	14	pure cultures	Garretson and SanClemente 1968
Total nitrogen	–				clay loam	Vicario 1972
phosphorus	–				clay loam	Ibid.
calcium	–				clay loam	Ibid.
pH of soil	0				clay loam	Ibid.
Nitrosomonas europaea	–	10 µg/ml	10		pure cultures	Garretson and SanClemente 1968
Fungi:						
Rhizoctonia solani	–				in vitro	[illegible] 1971
Monilia fructigena	–				in vitro	Ibid.
Colletotrichum lindemuthianum	–				in vitro	Ibid.
Phytophthora infestans	–				in vitro	Ibid.
Helminthosporium sativum	–				in vitro	Ibid.
Pythium debaryanum	–				in vitro	Ibid
Beauveria bassiana	–				liquid medium	Ramaraje *et al.* 1967
Metarrhizium anisopliae	–	0.5%	5000		liquid medium	Ibid.
Bacteria:						
Gram negative	–	5-100 µg/l	5-100			Newton 1972
Gram positive	–	5-100 µg/l	5-100			Ibid.

+ = stimulation   0 = no effect   – = inhibition

year after treatment of podzol soil with 2 lbs/A (2.24 kg/ha) of malathion (Hartenstein 1960), but MacPhee and Sanford (1956) described the effects of malathion on beneficial arthropods in Nova Scotia apple orchards as drastic.

The knock-out level of malathion for *Daphnia magna* was ten ppm for 19 minutes (Klimmer and Pfaff 1955), while the median tolerance limits for freshwater shrimp (*Gammarus lacustris*) was 0.00162 mg/l (0.00162 ppm) (Gaufin et al. 1965).

Fish and amphibians--Malathion is less toxic to fish than endrin or endosulfan (Toor et al. 1973). All rainbow trout (*Salmo gairdneri*) died when exposed to one mg/liter (one ppm); the 96 hour LC_{50} was 0.17 ppm (Pimentel 1971). In ponds, 0.5 lb/A (0.56 kg/ha) of malathion killed 68 percent of the mosquitofish (*Gambusia affinis*) which were placed in the water after 24 hours, and 70 percent of those placed in the water 48 hours after malathion treatment (Mulla and Isaak 1961). Emulsifiable concentrates were more toxic than technical grade malathion to four species of warm-water fish, but the difference was slight (Pickering et al. 1962).

Birds--The acute oral LD_{50} for young mallards was 1485 mg/kg, and the LC_{50} was over 5000 ppm when two week old birds were fed treated feed for five days followed by clean feed for three days (Pimentel 1971). Feeding of 0.1 ppm malathion decreased egg production in hens (Sauter and Steele 1972). Transient paralysis lasting for four to 14 days was observed in chickens given 100 mg/kg of malathion (Gaines 1969). In tissue culture, the ID_{50} (the dose causing 50 percent growth inhibition) of malathion on embryonic chick muscle was between ten and 100 ppm (Wilson et al. 1973). Malathion caused hypoglycemia and increased histogenesis of the islets of Langerhans in chicken embryos (Arsenault and Gibson 1974).

Injection of malathion into the yolks of hens' eggs caused malformations of legs and beaks (Walker 1971; Greenberg and LaHam 1969, 1970;

McLaughlin et al. 1963; Donachie and Fletcher 1969; Roger et al. 1969). Levels as low as 15 µM were effective, and cholinesterase inhibition was not the basis for teratogenesis, which could be prevented by nicotinamide (Walker 1971). Quinolinic acid, nicotinic acid, glycine, and tryptophan also decreased the incidence of malformations but only tryptophan prevented growth retardation (Greenberg and LaHam 1970).

Mammals--The inhibition of cholinesterase by malaoxon is minimal in mammals because of the rapid hydrolysis of malaoxon (Cook et al. 1958, Murphy et al. 1968, Seuterman and Main 1966). The acute oral LD$_{50}$ of malathion in rats is 1,000 mg/kg in females and 1,375 mg/kg in males (Gaines 1969); Jones et al. (1968) cited 1,400 and 1,900 mg/kg.

Among the reported effects of sublethal doses of malathion are hyperglycemia in rats given a subclinical dose of two to 2.5 mg/kg subcutaneously (Rams and Drexler 1973) and decreased nidation and increased early resorption in rats given malathion four to eight days after insemination (Lauro et al. 1969). In tissue culture, malathion at 200 to 400 µg/ml (200-400 ppm) inhibited mitoses (Huang 1973); growth of Chang liver cells could be inhibited by 13 µg/ml (13 ppm) (Gabliks and Friedman 1965). Malathion also inhibited the in vitro replication of vaccinia and poliomyelitis viruses (Gabliks 1967). In rats treated with 4,000 mg/kg (4,000 ppm) for two generations, the only adverse effect reported was an increase in ringtail disease in the second generation (Kalow and Marton 1961).

In humans, malathion is a moderately potent allergen, with allergies occurring under field conditions (Milby and Epstein 1964). Gantlov (1968) recommended a distance of 1,000 meters between areas of malathion production and human or animal habitations, because of the hazards of byproducts and waste products.

Conclusions:

The rapid degradation of malathion makes it eminently suitable for soil disposal because of its short persistence and minimal effects on soil processes.

Diazinon

Diazinon is the common name for *O,O*-diethyl *O*-6-methyl-2-(1-methylethyl)-4-pyrimidinyl phosphorodithioate, introduced by the Geigy Chemical Company in 1952 as Basudin. It is a nonsystemic insecticide with some acaricidal activity. Diazinon is prepared by the condensation of diethyl phosphorochloridothionate with 2-isopropyl-4-methyl pyrimidin-6-ol, which was in turn prepared by the condensation of isobutyramidine and ethyl acetoacetate (Martin 1968). It is a colorless oil with a boiling point of 83° to 84°C at 0.002 mm Hg and a vapor pressure of 1.4×10^{-4} mm Hg at 20°C. Its solubility in water is 40 ppm at room temperature and it is readily miscible with ethanol, acetone, and xylene. It is also soluble in petroleum oils. Technical diazinon is a dark brown liquid of approximately 95 percent purity.

Degradation

Biological--Diazinon is degraded in soil by a combination of microbial and chemical reactions. The initial step is the hydrolysis of the heterocyclic *P-O* bond, resulting in formation of diethylthiophosphoric acid and 2-isopropyl-6-methyl-4-hydroxypyrimidine (Getzin 1967, Lichtenstein et al. 1968, Bartsch 1973). The latter product is resistant to further degradation under anaerobic conditions, (Sethunathan 1972a, Sethunathan and Yoshida 1969, 1973a). Sethunathan and MacRae (1969) considered chemical hydrolysis to precede microbial action, but Sethunathan (1972a) subsequently reported greater persistence of diazinon in sterilized than in normal soil, and also noted increased hydrolysis of diazinon after repeated applications (Sethunathan 1972a, 1972b). Microbial degradation of diazinon is summarized in Table 71.

Tetraethylpyrophosphate was cited as one product of microbial diazinon degradation (Paris and Lewis 1973). A *Flavobacterium* species isolated

Table 71. DEGRADATION OF DIAZINON BY MICROORGANISMS AND IN SOIL

Conditions	Organism	Product	Reference
Irradiated soil		unidentified	Getzin and Rosefield 1968
Soil and water	unidentified	IMHP* and diethylthiophosphoric acid	Konrad *et al.* 1967
Soil, water, NaN_3		diethylthiophosphoric acid	Lichtenstein *et al.* 1968
Sterilized submerged soil		IMHP*	Sethunathan and Yoshida 1969
Natural submerged soil	unidentified	IMHP*	Ibid.
Soil	unidentified	IMHP*; CO_2	Getzin 1967
Rice paddies	unidentified	IMHP*; CO_2	Sethunathan 1972a
Paddy soil in water	heat labile, O_2-independent	water-soluble	Nasim *et al.* 1972
Laboratory	*Trichoderma viride*	unidentified	Matsumura and Boush 1968
Aerobic	*Flavobacterium*	CO_2	Sethunathan 1972a; Sethunathan and Yoshida 1973,1973b
8 species tested	2 microbial species	unidentified	Hirakoso 1969
	Arthrobacter, *Streptomyces*	CO_2	Gunner and Zuckerman 1968
Submerged soil	*Streptomyces*	CO_2	Sethunathan and MacRae 1969
Culture	*Pseudomonas melophthora*	26.7% water soluble	Boush and Matsumura 1967

*IMHP = 2-isopropyl-4-methyl-6-hydroxypyrimidine from paddy water was able to convert 30 percent of ^{14}C-labeled diazinon to carbon dioxide, with intermediate formation of 2-isopropyl-6-methyl-4-hydroxypyrimidine; diazinon itself was 95 percent depleted within 72 hours (Sethunathan and Yoshida 1973a). Diazinon was more rapidly degraded in sand than in loam, and more rapidly in moist than in dry soil; steam-treating the soil affected persistence more than soil type, moisture levels, or concentration (Bro-Rasmussen et al. 1968). Decreased diazinon degradation due to destruction of heat-labile soil substances was not observed by Getzin and Rosefield (1968). However, hydrolysis of diazinon to diethylthiophosphoric acid and 2-isopropyl-4-methyl-6-hydroxypyrimidine occurred readily in the presence of sodium azide (Lichtenstein et al. 1968).

When diazinon was applied to the surface of paddy soil, emulsions were more rapidly degraded than granules (Masuda and Fukada 1970). In a New York State muck soil, addition of marl (a finely powdered mixture of calcium carbonate and clay) speeded diazinon degradation in unsterilized soil, but retarded it in autoclaved soil (Kageyama et al. 1972). Nasim and co-workers (1972) reported that the degradation of diazinon in Dacca paddy field soil was carried out by unidentified, heat-labile substances; degradation reportedly proceeded for 24 hours and then stopped.

Among the specific organisms which have been found to degrade diazinon are *Arthrobacter* and *Streptomyces* acting together (Gunner and Zuckermann 1968); *Streptomyces* alone in the presence of glucose (Sethunathan and MacRae 1969); *Trichoderma viride* (Matsumura and Boush 1968) and *Flavobacterium* (Sethunathan and Yoshida 1972a, 1973a). The latter also degraded parathion, but not malathion (Sethunathan and Yoshida 1973a). Two of eight microbial colonies tested by Hirakoso (1969) were able to degrade diazinon. Microorganisms from sewage lagoons degraded diazinon less readily than either malathion or parathion (Halvorson et al. 1971). In mosquito-breeding polluted water, diazinon remained unaltered for days if the medium was alkaline (Hirakoso 1968). Diazinon was removed from culture by all planktonic algae tested, but no estimates of degradation were made (Butler *et al.* 1975).

Pseudomonas melophthora, the intestinal symbiote of the apple maggot, degraded diazinon to some extent, with 71 percent of the diazinon remaining unmetabolized, 26.7 percent converted to water-soluble substances, and 2.4 percent to solvent-soluble substances (Boush and Matsumura 1967).

In plants, metabolism was primarily due to hydrolysis of the pyrimidinylphosphorus ester bond with foliage the main site of hydrolysis. Accumulation of 2-isopropyl-4-methylpyrimidin-6-ol, and possibly of conjugated metabolites, occurred in the leaves (Kansouh and Hopkins 1968).

Rat liver microsomes were capable of degrading diazinon *in vitro* to diethylphosphorothioic acid and diethyl phosphoric acid (Yang *et al.* 1969); *in vivo*, rats excreted 50 percent of the radioactivity from ^{14}C-diazinon labeled at the pyrimidine and ethoxy moieties. No pyrimidine ring cleavage was detected (Muecke *et al.* 1970). Rats hydrolyzed the alkyl-*P* bond of diazinon to form diethylphosphoric acid at high levels of diazinon treatment. At lower levels, hydrolysis of the aryl-*P* bond was the primary degradative pathway (Plapp and Casida 1958). Sheep treated with diazinon excreted hydroxy-diazinon and its isomer, diethyl-6-hydroxymethyl-2-isopropyl-4-pyrimidinyl phosphorodithionate, in their urine (Machin *et al.* 1972). Dogs excreted 50 percent of the total radioactivity of ^{14}C-labeled diazinon in their urine within 24 hours; 16 percent as diethylphosphoric acid and 45 percent as diethylthiophosphoric acid (Iverson *et al.* 1975).

Chemical and physical--Hydrolysis of diazinon in soil was considered to be adsorption-catalyzed rather than acid-catalyzed, was more rapid in soil than in water, and was extremely rapid at pH 2, but slow at pH 6. Degradation was most rapid in Poygan sand (11 percent per day) and least rapid in Ella sediments (six percent per day); Kewaunee dolomitic till was intermediate. Hydrolysis was considered more significant than bacterial action in the degradation of the parent compound (Konrad *et al*. 1967). In water between pH 3.1 and pH 10.4, diazinon was hydrolyzed to 2-isopropyl-4-methyl-6-hydroxypyrimidine at different rates although first-order kinetics applied to all pH tested. Diazinon persisted for less than 145 hours at pH 10.4, over 3200 hours at pH 9.0, and over 4435 hours at pH 7.4 (Gomaa *et al*. 1969). It was reported that formulated diazinon degraded during storage to form tetraethylmonothiopyrophosphate (Mello *et al*. 1972).

Photolytic--Ultraviolet irradiation of diazinon results in formation of hydroxydiazinon (Paris and Lewis 1973). Photolytic degradation of diazinon was catalyzed by anthroquinone and to a lesser extent by pentachlorophenol (Ivie and Casida 1971).

Transport-

Within soil--The biological activity of diazinon in soil depends both on soil type and on the soil moisture. Diazinon is more toxic in mineral soil than in muck (Harris and Mazurek 1966a). It is more toxic in moist than in dry clay or sand (Harris 1966b) but the effect is more pronounced in sand; diazinon is somewhat more toxic in dry muck than wet muck (Harris 1964a, 1964b).

Diazinon was more mobile in brown forest soil than in either degraded chernozem or marsh soil, and was more mobile in soil than either phorate or lindane (Ostojic *et al*. 1972). The diffusion coefficient of diazinon in a silt loam was calculated to be 0.63 mm^2 per day at 27°C, and increased with increasing temperature, decreasing soil bulk density; and, to a lesser extent, with increasing moisture (Ritter *et al*. 1973). In 17.8 cm soil columns, diazinon was more mobile than triazine herbicides, chlorinated hydrocarbon insecticides, phorate, or disulfoton (Harris 1969a).

Between soil and water--Diazinon was completely eliminated from a model cranberry bog seven days after application (corresponding to six days after the bog was flooded); microflora were considered the main agents of degradation, but both fish and mussels accumulated diazinon. After 144 hours (six days), diazinon had been completely removed from the water (Miller et al. 1966). On a small agricultural watershed, the loss of diazinon in surface runoff or sediment was considered insignificant (Ritter et al. 1974).

Volatilization--Volatilization accounted for the loss of 86 percent of the diazinon on watch glasses over a 90 day period at 35°C. Loss of 77.5 percent occurred within the first 15 days (Alessandrini and Amormino 1954).

Into organisms--Diazinon was taken up by the roots of bean plants and translocated within plants. If the roots were subsequently rinsed and placed in nutrient solution, diazinon migrated from the roots into the nutrient solution (Kansouh and Hopkins 1968). Cabbages watered with two ppm of diazinon contained 0.01 ppm after 30 to 40 days, and tobacco contained 0.02 ppm after 56 days (Miles et al. 1967).

In spinach, diazinon accumulated at more than one ppm in the leaves, and at up to 60 ppm in the roots, when the soil was treated with 10 ppm; residues in strawberries remained below 0.1 ppm, and no diazinon residues were detected in onions even when soil levels were 100 ppm (Kono 1974). In rice paddies, diazinon applied to the water was absorbed by the leaf sheath of the plants whereas soil-applied diazinon was absorbed through the roots; in the latter case, none of the radioactivity was released into the water (Hirano and Yashima 1969). The maximum residues of diazinon in rice plants occurred five days after treatment (Sethunathan et al. 1971).

Emulsified ^{32}P-diazinon was more rapidly taken up into rice leaves from paddy soil than were granules. Residues of the former reached their highest levels in leaves within one day of treatment, while leaf residues after granule treatment rose continuously for 16 days after treatment (Hasuda and Fukada 1970). The major metabolites in rice leaves were $(C_2H_5O)_2P(O)H$, 50.4 percent; $(C_2H_5O)_2P(S)OH$, 31.1 percent; H_3PO_4 and $P(S)OH_3$, 7.2 percent. In rice ears, the major metabolite (85.9 percent) was $(C_2H_5O)P(O)OH$ (Hasuda and Fukada 1970).

Persistence:

Sterilizing soil by steam treatment reportedly increased the persistence of diazinon more than altering the type of soil, its moisture, or the level of diazinon applied (Bro-Rasmussen *et al.* 1968). Sethunathan (1972a) and Sethunathan and MacRae (1969) reported that sterilization increased the persistence of diazinon in all soils except acid clays (pH 4.7).

Decreasing the temperature of the soil from 35° to 15°C increased the persistence of diazinon by a factor of eight (Getzin 1968); in another study, reducing the soil temperature from 24° to 13°C lengthened the period of bioactivity of diazinon against a test insect (*Folsomia candida*) from less than six weeks to 16 weeks (Thompson 1973). Decreasing the soil moisture from 30 percent to 2 percent of field capacity increased the soil "half-life" of diazinon from six weeks to 16 weeks (Getzin 1968). The effects of soil type, temperature, moisture, and sterilization all interact in determining the persistence of diazinon (Bro-Rasmussen *et al.* 1968). At 90°F and 55 ± 5 percent relative humidity, bioactivity of diazinon reportedly persisted for 29 days (Kalkat *et al.* 1961).

Kageyama *et al.* (1972) reported that the addition of marl (a finely powdered mixture of calcium carbonate and clay) halved the persistence of diazinon in ordinary muck soil, but enhanced its persistence in sterilized muck soil. After seven months in sandy loam soil, one percent of two kg/ha diazinon remained, but ten percent remained in peaty loam (Suett 1971). Diazinon residues of 13 percent remained in henhouse litter after four weeks, (Galley 1972) and 4.6 percent remained in neutral aquerus medium after five weeks (Cowart *et al.* 1971).

Under ordinary agricultural conditions, the biological persistence of diazinon ranges between two and four weeks (Krishnan *et al.* 1968, Getzin and Rosefield 1966, Bro-Rasmussen *et al.* 1968, Harris 1969b, Harris and Hitchon 1970). Read (1969) reported that the effectiveness of diazinon in mineral soil decreased steadily for one month and then remained nearly unchanged for during the second and third month. In a newly developed irrigation district, foliar sprays of diazinon left initial residues of up to 3.34 ppm in the soil, which decreased to 0.02-0.05 ppm within 1.5 to 2.5 months (Knutson *et al.* 1971). When two to four times the normal agricultural levels of diazinon were applied to a carrot field, less than 0.3 ppm remained in the soil 42 days later (Stobwasser 1963), and when 400 g/A (about 0.45 ppm) were sprayed on field-grown lettuce, somewhat less than 0.1 ppm (22 percent) were detectable after ten days (Coffin and McKinley 1964).

In rice, diazinon was detected at 0.006 ppm 53 days after treatment with 2.5 kg/ha (Masuda and Kanazawa 1972). When soil was treated with 6 ppm, diazinon residues in the plant *Impatiens balsami* were detectable for 63 days (Augustinsson and Johnsson 1957).

Data on the persistence of diazinon are summarized in Table 72 for those studies in which the residue remaining after a specific length of time could be calculated. In summary, diazinon is most persistent if incorporated at high levels into a cold, dry, alkaline soil which has never before been exposed to diazinon or parathion. It is least persistent if sprayed at low levels onto a warm, moist, acid soil which has been repeatedly treated with diazinon. Under the former conditions, diazinon might be expected to persist well over three months, while under the latter conditions it would be dissipated, if not degraded, within two to three weeks.

Table 72. PERSISTENCE OF DIAZINON IN SOIL

% residue	# days	% mean loss/day	Conditions	Reference
0	>180	0.56	neutral, unflooded soil	Sethunathan 1972a
10	168	0.06	field conditions	Getzin and Rosefield 1968
<8	140	>0.06	4 soils	Ibid.
10–15	140	<0.11	radioactivity measured	Getzin 1967
0	133	0.75	15 lbs/A; greenhouse; bioassay	Ahmed and Morrison 1972
100	>112	–	13°C, soil, bioassay	Thompson 1973
0	98	1.02	15 lbs/A; field; bioassay	Ahmed and Morrison 1972
3	91	1.10	10 lbs/A, field, bioassay	Ibid.
0	91	1.10	10 lbs/A; greenhouse; bioassay	Ibid.
0	>70	<1.43	muck; bioassay	Kageyama et al. 1972
2–6	50–70	1.34–1.96	submerged soils	Sethunathan and MacRae 1969
0	63	1.59	5 lbs/A; field	Ahmed and Morrison 1972
0	60	1.67	neutral, flooded soil	Sethunathan 1972a
13	53	0.25	granules; soil	Masuda and Kanazawa 1972

Table 72. Cont'd

% residue	# days	% mean loss/day	Conditions	Reference
.0065	50	2.00	2 lbs/A, pasture	VanMiddelem et al. 1972
0	49	2.04	5 lbs/A; greenhouse; bioassay	Ahmed and Morrison 1972
0	29	3.45	bioassay	Kallkat et al. 1961
50	25	2.00	soil	Stathopoulos et al. 1971
.02	21	4.76	2 lbs/A, pasture	VanMiddelem et al. 1972
0	16.7	6.00	Ella fine sediments	Konrad et al. 1967
0	14.3	7.00	Kewaunee dolomitic till	Ibid.
0	9.1	11.00	Poygan sand	Ibid.
0	9	11.11	sprayed on soil; bioassay	Laygo and Schulz 1963

Effects on Non-Target Species:

Microorganisms--Diazinon was not mutagenic in *Escherichia coli* under conditions in which several organophosphorus compounds were mutagenic (Mohn 1973). Diazinon, but not its metabolites diazinon-oxon, diethylphosphorothioate, and 2-isopropyl-4-methyl-6-hydroxyprimidine, markedly inhibited the growth of heterotrophic aerobic bacteria (Robson and Gunner 1970). At 40 to 120 kg/ha, diazinon formulated as Basudin inhibited nodule formation in red clover (*Trifolium pratense*) grown on sandy soil. Nodule formation was also inhibited in alfalfa (*Medicago sativa*) grown on chernozem. If applied at 80 to 120 kg/ha, diazinon decreased the nitrogen content of the clover (Salem et al. 1971).

At 100 µg/g (100 ppm), diazinon stimulated fungal growth for one week. Ammonification increased and nitrification decreased, but overall soil respiration was not affected (Tu 1970). At high levels, diazinon inhibited mycelial growth in the fungus *Aspergillus* (Eder 1963), but neither the growth nor the carbon-14 assimilation of the freshwater alga *Scenedesmus quadricauda*tus was affected by diazinon in the culture medium (Stadnyk et al. 1971).

The data on the effects of diazinon on soil microorganisms are summarized in Table 73. It must be noted that all applications showing adverse effects are well above ordinary agricultural levels of application.

Invertebrates--Diazinon, sprayed on white clover (*Trifolium repens*) at 1/8 pound per acre (0.14 kg/ha) of a 40 percent wettable powder caused 28 percent mortality to bees present on the clover; residual toxicity after three hours was 78 to 92 percent mortality if 1/4 pound per acre (0.28 kg/ha) was sprayed (Clinch 1969). In soil, diazinon decreased the numbers of parasitic mites and of paurapods; the numbers of springtails increased (Edwards et al. 1969). Increases in the numbers of springtails were secondary to decreases in the numbers of predatory

Table 73. EFFECTS OF DIAZINON ON SOIL MICROORGANISMS AND SOIL PROCESSES

Parameter	Effect	Level Original	 PPM	observed	Soil or conditions	Reference
Soil respiration	-	100 μg/g	100	21	sandy loam; laboratory	In 1970
Nitrification	-	100 μg/g	100	21	sandy loam; laboratory	Ibid.
Fungi	+	100 μg/g	100	7	sandy loam; laboratory	Ibid.
	0	100 μg/g	100	8-56	sandy loam; laboratory	Ibid.
Nodule formation in legumes	-	40-120 kg/ha	~20-60	growing season	sandy soil or chernozem	Salem *et al.* 1971
Heterotrophic, aerobic bacteria	-				culture	Bohnen and Gunner 1970
Scenedesmus (alga)	0				culture	Stadnyk *et al.* 1971

+ = stimulation
0 = no effect
- = inhibition

mites (Edwards and Thompson 1973). In an old-field ecosystem, species diversity and density increased in the year following treatment with 14 lbs/A (15.7 kg/ha) of diazinon (Malone et al. 1967).

Plants--Diazinon at 14 lbs/A (15.7 kg/ha) increased root growth and decreased shoot growth in an old-field ecosystem; the number and density of plant species increased for up to one year after treatment (Malone et al. 1967).

High levels of diazinon reportedly inhibited both shoot and root growth in higher plants (Eder 1963), but Dennis and Edwards (1963) did not consider diazinon phytotoxic.

Fish and amphibians--Diazinon was not toxic to young rainbow trout (*Salmo irideus*) at 0.1 mg/liter (100 ppm), caused some deaths at 0.4 mg/liter (400 ppm), and killed all the trout at 0.5 mg/liter (500 ppm). Pike (*Esox lucius*) were somewhat less sensitive, with the first deaths occurring at 0.5 mg/liter (500 ppm) and all the pike killed at two mg/liter (2000 ppm) (Luedemann and Neumann 1961). The 48 hour EC_{50} (effective median concentration) of diazinon was 170 ppb in rainbow trout and 86 ppb in bluegills at $24^{0}C$ (Pimentel 1971). The LD_{50} for bullfrogs was over 2000 mg/kg (Tucker and Crabtree 1970).

Birds--The oral LD_{50} of diazinon was 3.5 mg/kg in young mallards and 4.3 mg/kg in pheasants (Tucker and Crabtree 1970). For redwinged blackbirds and starlings, the acute oral LD_{50} was 110 mg/kg and 2.0 mg/kg respectively (Schafer 1972). The five day dietary LC_{50} in bobwhites, pheasants, Japanese quail, and mallards was 245 ppm, 244 ppm, 47 ppm, and 191 ppm, respectively (Heath et al. 1972). Diazinon was teratogenic in chicks (Green 1970) and reduced hatchability at levels of one ppm (Sauter and Steel 1972); egg production declined at 180 ppm (Vondell 1958).

Mammals--The acute oral LD_{50} of diazinon in rats is variously given as 75 mg/kg in females and 108 mg/kg in males (Gaines 1968, Pimentel 1971); 150 to 220 mg/kg (Schafer 1972) or even 300 to 600 mg/kg (Jones et al.

1968). Formulated diazinon which had decomposed during storage was 30 times as toxic to humans and to cattle as freshly formulated diazinon because of the formation of tetraethylmonothiopyrophosphate (Meier *et al.* 1979). Like all organophosphates, the oxon of diazinon is a cholinesterase inhibitor; its toxicology was described by Gysin and Margot (1958) and the toxicology of organophosphates including diazinon has been reviewed recently (Eto 1974).

At 100 to 200 mg/kg, diazinon was both toxic to pregnant rats and teratogenic to their fetuses (Kimbrough and Gaines 1968). Seven mg/kg and 30 mg/kg of diazinon did not produce congenital malformations in rabbits, and 0.125 and 0.25 mg/kg were not teratogenic in hamsters (Robens 1969). A mutagenic effect of diazinon on human peripheral leukocytes in culture has been reported (Tsoneva-Maneva *et al.* 1969).

Conclusions:

Neither the persistence of diazinon itself nor its effects on soil processes and soil microorganisms is extreme. Nevertheless, there is no conclusive evidence for the decomposition of diazinon to naturally occurring substances, and data are lacking on the persistence, transport and effects of 2-isopropyl-4-methyl-6-hydroxypyrimidine in soil. Therefore, large-scale soil disposal of diazinon in the absence of further information is not recommended.

Disulfoton and Phorate

Disulfoton is the common name for *O,O*-diethyl *S*-2-(ethylthio)ethylphosphorodithioate, introduced by Farbenfabriken Bayer AG in 1956 under the trade names Di-Syston and Dithio-Systox as a systemic insecticide and acaricide. It is made by the interaction of the sodium salt of *O,O*-diethyl hydrogen phosphorodithioate with β-chloroethyl thioethyl ether (Martin 1968). The pure compound is a colorless oil with a characteristic odor, a vapor pressure of 1.8 mm mercury at 20°C, and a water solubility of 25 ppm at 20°C. Its boiling point is 62°C at 0.01 mm mercury, and it is readily soluble in most organic solvents. The technical product is a dark yellow oil. Formulations include granules and impregnation on activated carbon.

Phorate is the common name for *O,O*-diethyl *S*-(ethylthio)methyl phosphorodithioate, introduced by the American Cyanamid Company as Thimet in 1954. It is a systemic insecticide used primarily to protect seedlings from sap-feeding insects. Phorate is made by the reaction of *O,O*-diethyl hydrogen phosphorodithioate with formaldehyde, followed by the addition of ethyl mercaptan. It is a clear liquid with a boiling point of 118 to 120°C at 0.8 mm mercury, a freezing point below -15°C, and a vapor pressure of 8.4×10^{-4} mm mercury at 20°C. Its solubility in water is 50 ppm at room temperature and it is readily miscible with vegetable oils and xylene, as well as with carbon tetrachloride and dioxane. It is formulated as an emulsifiable concentrate, dusts and granules.

Degradation:

Biological--Data on metabolic products of disulfoton and phorate are summarized in Table 74. The primary degradative pathways of disulfoton and phorate proceed via their sulfoxides to their sulfones, leading to an increase in the anticholinesterase activity (Metcalf et al.

Table 74. DEGRADATION OF PHORATE AND DISULFOTON

Pesticide	Organism(s) or conditions	Products	Reference
Phorate		sulfoxide, sulfone	Metcalf et al. 1957
	soil: Sultan silt loam	sulfoxide, sulfone	Getzin and Shanks 1970
	soil: 1,5,10 lbs/A, granules	sulfoxide, sulfone	Schulz et al. 1973
	soil: in ecosystem	sulfoxide, sulfone	Lichtenstein et al. 1974
	corn: in ecosystem	sulfoxide, sulfone	Ibid.
Disulfoton		sulfoxide, sulfone	Metcalf et al. 1957
	soil: paddy or upland	sulfoxide, sulfone	Takase et al. 1972
			Takase and Nakamura 1974
	soil	sulfoxide, sulfone	[illegible] and [illegible] 1972
	rats: liver, at 30 minutes	sulfoxide, sulfone	Bull 1965
	excreted	diethyl phosphorothioate, phosphoric acid, diethyl phosphorodithioate	Ibid.

1957, Fukuto and Metcalf 1969). Getzin and Shanks (1970) analyzed the degradation of phorate and its oxygen analog in soil, and noted that phorate is converted to phorate sulfoxide and then to its sulfone; in Sultan silt loam, conversion of phorate sulfoxide to phorate was also observed. In a plant-soil model ecosystem, corn plants contained only metabolites of phorate even though the soil still contained unaltered phorate. Corn roots as well as soil contained phorate sulfone and phorate sulfoxide, while the leaves of the plants contained phoratoxon sulfoxide and phoratoxon sulfone (Lichtenstein *et al.* 1974). When phorate was applied to soil in granular bands under field conditions, the major metabolite was phorate sulfone when the level of treatment was five lbs/A (5.60 kg/ha), but phorate sulfoxide when the level of treatment was one lb/A (1.12 kg/ha). The oxygen analog of phorate was not found in submerged soils (Kawamori *et al.* 1971b).

Almost no degradation of either phorate or disulfoton occurred in soil in a 60 day period in summer, and degradation was significantly greater between October and April than in the summer; temperature played a greater role in determining the rate of degradation than did soil type. In the winter, disulfoton was degraded more rapidly than phorate; in summer, too little degradation took place to distinguish differences. The soil type and temperature effects interacted, with degradation being more rapid in loamy sand in the winter and in silt loam in the summer (Menzer *et al.* 1970). Takase and co-workers (1972) examined the degradation of disulfoton in several soils. Metabolism was more rapid in all soils under flooded (anaerobic) than under upland (aerobic) conditions, microbial oxidation was considered the major route of degradation, and little breakdown occurred in sterile soil. A later study of the breakdown of disulfoton in sterile and glucose-amended soil was considered to provide evidence against microbial oxidation (Takase and Nakamura 1974).

Whether the most rapid route of degradation in the field is chemical or biological has never been proven, but disulfoton is readily degraded by microorganisms, at least under laboratory conditions: most fungi (among twenty isolates tested) were able to use disulfoton as their sole source of carbon, as were most of the ten cultures of Streptomycetes tested. *Aspergillus flavus*, *Helminthosporium*, and two *Streptomyces* species were able to grow with disulfoton as their only source of carbon (Bhaskaran *et al*. 1973).

In rats, the excreted metabolites of disulfoton were diethylphosphorothioic acid, diethyl phosphoric acid, and two unknown compounds, while phosphoric acid and traces of diethylphosphorodithioic acid were found in the rats' urine. Intermediates in the rats' livers, 30 minutes after intraperitoneal injection of 10.5 mg/kg of disulfoton, included disulfoton sulfoxide and sulfone (Bull 1965).

Chemical and physical--Di-Syston applied to fertilizer, particularly superphosphates, decomposes as a result of catalytic oxidation (Ibrahim *et al*. 1969). When disulfoton and phorate were exposed to one to four Mrad of cobalt-60 *gamma*-irradiation, the corresponding sulfoxides and sulfones were found in most samples; the sulfoxide of the oxygen analog and the sulfone of the oxygen analog were found only after treatment with the full four Mrad dose. Irradiation increased the inhibition of beef liver carboxylesterases by the pesticides, suggesting that the radiation-induced degradation consisted mostly of activation (Grant *et al*. 1969).

Transport-

Within soil--Phorate was less mobile than diazinon, and less mobile in degraded chernozem and in black marsh soil than in brown forest soil (Ostojic *et al*. 1972). McCarty and King (1966) considered disulfoton to move rapidly in soil-water systems. Harris (1969b) compared the mobility of pesticides in 17.8 cm soil columns and found phorate and disulfoton to be only slightly mobile, albeit more mobile than the chlorinated hydrocarbon insecticides. No data were available on the transport of the metabolites of disulfoton or phorate within soil.

Between soil and water--In addition to transport through soil into water, transport on soil particles (washoff) can be a means of pesticide transport into streams and lakes. Kawamori and co-workers (1971a) noted that disulfoton adsorbs more readily to silty clay loam than to clay loam, and least readily to alluvial loamy sand. They concluded that the high rate of recovery was due to adsorption on the organic rather than on the clay fraction. Both compounds were most readily adsorbed to mineral soils (Harris and Hitchon 1970) and to silty loam and clay soils (Bhirud and Pitre 1972) when the soils were dry. Soil-adsorbed disulfoton could not be desorbed if soil had dried in the interim (Graham-Bryce 1967). Transport of newly desorbed disulfoton or phorate in runoff remains a possibility, but no data are available to assess its plausibility.

Volatilization--Burns (1971) considered the 41 percent loss of phorate from sandy soil within the first three days after application to be due largely to volatilization. Losses from clays and loams were much smaller.

Into organisms--Both phorate and disulfoton are systemic insecticides, readily taken up by plants. Soil treated with two pounds per acre (2.24 kg/ha) of either phorate or disulfoton resulted in excessively high levels in spinach after 5.5 months; both parent compounds, their sulfoxides, their sulfones, and the sulfoxides of their oxygen analogs were found in the spinach (Menzer and Dittman 1968). When soil was treated with 30 kg/ha of five percent disulfoton granules (Solvirex) 17 days after potatoes were planted, potato tubers contained 0.04 to 0.11 ppm at harvest time (Trojanowski *et al.* 1967). Masuda and Kanazawa (1972) detected 0.18 ppm disulfoton in unpolished rice 53 days after soil was treated with 2.5 kg/ha. The residual levels of disulfoton in vegetables grown on treated soil were greatest in carrots, intermediate in chinese cabbage, and lowest in turnips. Carrots, rice, and soil contained relatively more disulfoton sulfone than disulfoton sulfoxide (Masuda and Kanazawa 1972).

When phorate was applied to soil or sand, only metabolites were found in the corn grown on the soil; oxidation appeared to occur in the plant, not in the soil. Root residues were 2.5 to five times as great in plants grown on sand as in plants grown on agricultural soils, but leaf residues were the same regardless of the soil type (Lichtenstein et al. 1974). Sorghum retained no residues of disulfoton 85 days after soil treatment (Singh et al. 1972), but carrots contained phorate sulfone even in the second growing season after soil treatment (Lichtenstein et al. 1973). Potatoes contained residues only in the growing season during which soil had been treated (Ratora et al. 1967).

Persistence:

The available data on persistence of disulfoton and phorate in soil are summarized in Tables 75 and 76. Persistence differs widely not only because of differing test conditions between experiments, but also because different assays were used. Bioassays dominate the literature on phorate and disulfoton; the data are therefore skewed to include insecticidal metabolites of the applied insecticides. Persistent but non-insecticidal metabolites have not been considered to any great extent in studies available to date. Among the possible factors decreasing the measured persistence of phorate and disulfoton are losses due to volatilization of sprayed insecticides and adsorption to soil. The latter almost certainly serves to prolong soil retention even while decreasing persistence as measured by bioassays.

Effects on Non-Target Species:

Microorganisms--The effects of phorate and disulfoton on soil microorganisms and soil invertebrates are summarized in Tables 77 and 78.

Cowley and Lichtenstein (1970) observed inhibition in 13 of 17 fungal cultures treated with 40 ug/ml (40 ppm) of phorate, but in a fescue meadow treated with agricultural levels of phorate, both bacteria and fungi were stimulated for most of one year (Malone and Reichle 1973).

Table 75. PERSISTENCE OF DISULFOTON IN SOIL

% Residue	# Of days	Mean loss/day %	Conditions	Reference
0	85	>1.18	granules in furrow	Singh *et al.* 1972
50	50	1.00	residues include metabolites	Takase and Nakamura 1974

Table 76. PERSISTENCE OF PHORATE IN SOIL

% residue	# of days	% mean loss/day	Conditions	Reference
trace	730	<0.14	250 ppm; acid-sandy soil	Way and Scopes 1968
10	540	0.17	250 ppm; bioassay	Way and Scopes 1965
5	100	0.95	bioassay	Read 1969
50	75	0.67	10 ppm; bioassay	Way and Scopes 1965
50	68	0.74	10 ppm, acid-sandy soil	Way and Scopes 1968
82	48	0.38	muck	Burns 1971
77	48	0.48	peat	Ibid.
66	48	0.71	peat-sand mixture	Ibid.
53	48	0.98	sand	Ibid.
24	48	1.58	clay	Ibid.
0	30	>3.33	bioassay	Parker and Dewey 1965
50	30	1.67	soil-incorporated, 4-5 inches	Lichtenstein *et al.* 1973
21	28	2.82	300 ppm, henhouse litter	Galley 1972
0	14-28	7.3-14.6	bioassay, sandy loam soil	Harris 1969b
50	6	8.33	soil-surface application	Lichtenstein *et al.* 1973
59	3	13.67	sand	Burns 1971
72	3	9.33	clay	Ibid.
99	3	0.33	muck	Ibid.
86	3	4.67	peat	Ibid.
74	3	8.67	peat-sand mixture	Ibid.

Table 77. EFFECTS OF DISULFOTON ON SOIL MICROORGANISMS AND SOIL INVERTEBRATES

Parameter	Effect	Level of treatment Original	ppm	# days Observed	Soil or conditions	Reference
Nitrate levels	+	60 kg/ha		50	volcanic ash soil	Kobayashi and Katsura 1968
Nitrifying bacteria,						
aerobic	–	high	–			Mahmoud et al. 1972
aerobic	+	low	–			Ibid.
anaerobic	–	high	–			Ibid.
Actinomycetes	–	high	–			Ibid.
Springtails	+					Edwards et al. 1968

+ = stimulation
0 = no effect
– = inhibition

Table 78. EFFECTS OF PHORATE ON SOIL MICROORGANISMS AND SOIL INVERTEBRATES

Parameter	Effect	Level of treatment Original	PPM	# days Observed	Soil or conditions	Reference
Bacteria	+			365	Fescue meadow	Malone and Reichle 1973
Fungi	+			365	Fescue meadow	Ibid.
Fungi (13 of 17)	–	40 µg/ml			Laboratory	Cowley and Lichtenstein 1970
Rhizoctonia solani	–				27°–33°C	Rao 1975
Fusarium moniliforme	–				Low temperatures	Ibid.
Collembola	–			365	Fescue meadow	Malone and Reichle 1973
Springtails	+					Edwards *et al.* 1969
Pauropods	–					Ibid.
Earthworms	–					Ibid.

+ = stimulation
0 = no effect
– = inhibition

The latter report suggests that phorate decreased the populations of soil arthropods, with secondary increases in bacteria and fungi.

Sreenivasulu and Rangaswami (1973) reported the stimulation of almost all soil microorganisms by phorate and disulfoton, but noted that, as the incubation progressed, gram-positive bacteria were displaced by gram-negative bacteria. Laygo and Schulz (1963) reported recovery of soil microfaunal populations within nine days of phorate application even though the compound itself persisted more than 23 days.

Invertebrates--Phorate decreased populations of *Collembola* and of soil arthropods other than mites (Malone and Reichle 1973), and decreased total numbers of mites for more than nine months if applied at 4.5 kg/ha (Edwards and Thompson 1973). Organophosphorus insecticides reduced the number and biomass of soil invertebrates less than did aldrin or dieldrin, but phorate and disulfoton significantly decreased populations of parasitic mites and paurapods, and increased populations of trombidiform mites, oribatid mites, and springtails. Phorate, but not disulfoton, significantly decreased earthworm populations (Edwards et al. 1969, Edwards and Thompson 1973).

The LD_{50} of phorate to bees was 0.004 percent (40 ppm) (Johansen 1961). The nectar of fuchsia and nasturtiums watered with 25 mg phorate was not toxic to honeybees and the acute oral LD_{50} of disulfoton and phorate to honeybees was reported as greater than 20 ug/bee and 0.44 ug/bee, respectively (Lord et al. 1968).

Disulfoton was toxic to aquatic insects at levels between 0.0082 mg/liter (stone fly, *Acroneuria pacifica*) and 0.24 mg/liter (freshwater shrimp, *Gammarus lacustris*) (Gaufin et al. 1965). Disulfoton decreased survival and affected development of the eggs of both oysters (*Crassostrea virginica*) and clams (*Mercenaria mercenaria*) at levels of one ppm or more (Davis and Hidu 1969).

Plants--At 60 kg/ha, disulfoton caused an initial delay in plant growth, followed by enhanced growth after 30 days (Kobayashi and Katsura 1968). At ten ppm, soil-incorporated disulfoton stimulated growth of bean and corn plants for six weeks, but at 100 ppm, growth inhibition occurred. Disulfoton at 100 ppm also increased the levels of nitrogen, phosphorus, potassium, calcium, and magnesium; and decreased levels of iron, copper, aluminum and zinc in the treated plants (Cole et al. 1968). Disulfoton inhibited the metabolism of linuron and dicamba in leafy tissues (Chang et al. 1971). Phytotoxicity between dalapon and either disulfoton or phorate was additive, indicating no interaction between the herbicide and either insecticide (Nash 1967).

Fish and amphibians--Phorate at agricultural levels was toxic to carp (Lilly et al. 1969). The 96 hour median tolerance limit (TL_m) for disulfoton was 0.063 ppm in bluegills (*Lepomis macrochirus*), 0.25 ppm in guppies (*Lebistes reticulatus*), 3.7 ppm in fathead minnows (*Pimephales promelas*), and 6.5 ppm in goldfish (*Carassius auratus*) (Pickering et al. 1962).

Birds--The acute oral LD_{50} of disulfoton was greater than 32 mg/kg in starlings, and was 3.2 mg/kg in redwinged blackbirds; for phorate, the LD_{50} was 7.5 mg/kg in starlings and 1.0 mg/kg in redwinged blackbirds (Schafer 1972). The dietary LC_{50} of disulfoton when birds were fed treated feed for three days was 333 ppm in Japanese quail (*Coturnix coturnix*), 715 ppm in bobwhite (*Colinus virginianus*), 634 ppm in pheasants, and 510 ppm in mallards (*Anas platyrhynchos*) (Heath et al. 1972).

Phorate (as Thimet) injected into hens' eggs at two ppm on the 10th day of incubation decreased the hatchability from 89.7 to 27.5 percent, increased the thyroid size of the chicks, and was teratogenic (Richert and Prahlad 1972).

Mammals--The acute oral LD_{50} of phorate in rats was reported to be two to three mg/kg (Jones et al. 1968); for disulfoton, an LD_{50} of 12.5

mg/kg is cited by Pimentel (1971), while Jones *et al.* (1968) cited 6 mg/kg. Both compounds were toxic when applied to rats' skins, with a dermal LD_{50} of 70 to 300 mg/kg cited for phorate and 50 mg/kg for disulfoton (Jones *et al.* 1968).

Conclusions:

Most of the data on the persistence of phorate and disulfoton in soil are based on persistence of bioactivity, rather than on chemical persistence. Moreover, essentially no data on the transport of either compound in soil, into water, or into air are available. Therefore, no conclusions on the feasibility of soil disposal of either phorate or disulfoton can be drawn.

Azinphosmethyl

Azinphosmethyl is the common name for *O,O*-dimethyl *S*-(4-oxo-1,2,3-benzo-triazin-3(4H)-yl)methyl phosphorodithioate. It is made by the interaction of *N*-chloromethylbenzazimide with *O,O*-dimethyl hydrogen phosphorodithioate in the presence of a base. A non-systemic insecticide and acaricide, azinphosmethyl was introduced by Farbenfabriken Bayer AG in 1953 and is known under the trade names Guthion and Gusathion. Azinphosmethyl forms white crystals with a melting point of 73 to 74°C. It is very slightly soluble in water (3.3 ppm at 25°C) and is soluble in most organic solvents. It is unstable above 200°C.

Degradation—

Biological—Microbial degradation of azinphosmethyl was presumed to account for the faster loss of azinphosmethyl from natural than from sterilized soil, but non-biological factors appeared to predominate (Yaron *et al.* 1974a, see below). In mice, the microsomal and soluble fractions of liver cells were most active in converting azinphosmethyl to its oxygen analog, and also in degrading it to dimethyl phosphoric acid and dimethyl phosphorothioic acid (Motoyama 1972).

Chemical—Yaron and co-workers (1974a) analyzed the kinetics of azinphosmethyl loss from soil and concluded that both biological and chemical mechanisms contributed to its degradation. A lag phase was observed in all soils; subsequent degradation essentially corresponded to first-order kinetics. Degradation was most rapid, and the lag phase least pronounced, in moist, unsterilized soil at warm temperatures. Table 79 shows the length of time required for the loss of 50 percent of the azinphosmethyl applied to a silty, loamy, loessial sierozem. Moisture affected the rate of degradation more than did microbial activity, and temperature was extremely important in determining the half-life of azinphosmethyl within each soil/moisture combination.

Table 79. NUMBER OF DAYS REQUIRED FOR 50% LOSS OF AZINPHOSMETHYL FROM SOIL AT THREE TEMPERATURES AND TWO LEVELS OF MOISTURE (AFTER YARON *ET AL.* 1974a)

Temperature (°C)	Sterile soil		Natural soil	
	Dry	Wet	Dry	Wet
6	484	88	484	64
25	135	29	88	13
40	36	6	32	5

Photolytic--Ultraviolet light at 2537 Å decomposed azinphosmethyl labeled at the carboxyl carbon. The degradation products were: benzazimide, anthranilic acid, methyl benzazimide sulfide, N-methyl benzazimide, and an unidentified water-soluble compound. None of the products was insecticidal. Degradation proceeded only in the light, and was more rapid in water at high pH than on glass. In water at pH 10, 18 percent of the metabolites were water-soluble, while at pH 11, 97 percent were water-soluble. Azinphosmethyl was stable in water between pH 6 and pH 9 (Liang and Lichtenstein 1972).

Transport:

Through soil--Eight years after an undiluted (18.1 percent) emulsifiable concentrate of azinphosmethyl was applied to soil, no residues were detected below 37.5 cm in sandy loam soil (Staiff et al. 1975). Yaron et al. (1974b) concluded that azinphosmethyl did not leach deeply into soil, and also that its leaching was unaffected by the amount of irrigation.

Volatilization--Azinphosmethyl was less readily volatilized from leaves or from soil than were diazinon, parathion, and several organochlorine insecticides (Lichtenstein and Schulz 1970). Its volatilization from leaves was reportedly not affected by high temperatures (Hightower and Martin 1958).

Into organisms--Azinphosmethyl applied to an irrigated field at normal agricultural levels resulted in residues in potato peels (Yaron et al. 1974b).

Persistence:

Staiff and co-workers (1975) assessed the persistence of several formulations and several levels of azinphosmethyl in sandy loam soil over an eight year period. The soil pH ranged from 6.6 to 7.8, and rainfall averaged 25 cm per year. Their data are summarized in Table 80. They concluded that soil contamination by undiluted azinphosmethyl remained

Table 80. RESIDUES OF AZINPHOSMETHYL IN SOIL AFTER CONTAMINATION WITH UNDILUTED (18.1%) AND DILUTED (0.045%) EMULSIFIED CONCENTRATE SOLUTIONS AND DILUTED SOLUTIONS (0.045%) OF WETTABLE POWDER.

Contaminant; soil stratum	Time after application 1 day	 6 years	 8 years
EC[a]; 18.1%;			
0-2.5 cm	49,946 ppm	6,075 ppm	850 ppm
2.5-7.5 cm	30,488 ppm	7,862 ppm	967 ppm
EC[a]; 0.045%			
0-2.5 cm	354 ppm	1.5 ppm	ND[b]
2.5-7.5	23 ppm	1.3 ppm	ND[b]
WP[c]; 0.045%			
0-2.5 cm	276.8 ppm	3.0 ppm	ND
2.5-7.5 cm	107.0 ppm	2.2 ppm	ND

a/ Emulsifiable concentrate

b/ Not detected

c/ Wettable powder

a significant hazard for at least four years.

The soil residues in the top 7.5 centimeters four years after the soil was contaminated with diluted azinphosmethyl at 0.045 percent (450 ppm) represented 1.6 percent of the initial residues, or a loss of 24.6 percent per year while the undiluted emulsifiable concentrate left residues representing 2.5 percent of the initial residues after eight years, corresponding to a loss of 12.2 percent per year. It was suggested that high concentrations of azinphosmethyl strongly inhibit soil microorganisms, resulting in slower degradation of the pesticide.

Soil-applied azinphosmethyl decomposed rapidly in Georgia cotton-growing soil, but traces could be detected to the end of the third year (Roberts *et al*. 1962). At agricultural levels in an irrigated field, azinphosmethyl reportedly disappeared within thirty days regardless of the amount of irrigation (Yaron *et al*. 1974). Kuhr *et al*. (1974) also reported that three lbs/A (3.36 kg/ha or 6 ppm) of azinphosmethyl decreased to 1.6 ppm within thirty days in one orchard; essentially complete disappearance occurred within sixty days in a second orchard.

In water, azinphosmethyl has a half-life of about thirty days at pH 9.0, but of less than seven days at pH 9.5, when the temperature is at 35°C; at 45°C and pH 9.0, the half-life decreased to less than one day (Heuer *et al*. 1974). In comparing the persistence of water emulsion sprays with that of low-volume sprays on oat plants, Dorough and Randolph (1967) found that azinphosmethyl persisted longer as a low-volume spray (21 days) than as a water emulsion spray (Seven days).

Effects on Non-Target Species:

Microorganisms--Azinphosmethyl reportedly had no effect on yeast respiration of fermentation at concentrations ranging between 2×10^{-5} M and 2×10^{-3}M (Eder 1963). Staiff *et al*. (1975) considered it probable that azinphosmethyl at high levels partially sterilized the soil, hindering its own degradation.

Plants--No reports of phytotoxicity were found for azinphosmethyl, but it has been reported to inhibit photosynthesis in the leaves of Red Delicious apples (Heinicke and Foot 1966).

Invertebrates--Azinphosmethyl is more highly toxic to earthworms (*Eisenia* sp.) than malathion or the organochlorines BHC, chlordane, heptachlor, aldrin and dieldrin (Hopkins and Kirk 1957). It is highly toxic to honeybees (Anderson and Atkins 1958, Anonymous 1974). When ladybird beetles were immersed in azinphosmethyl at concentrations of 0.5 lb/100 gallons, 35 to 65 percent survived (Colburn and Asquith 1973).

Fish and amphibians--The 96 hour TL_M of young salmonids to azinphosmethyl was 0.58 ppb (0.0006 ppm) for rainbow trout, 4.2 ppb (0.0042 ppm) for coho salmon, and 4.3 ppb (0.0043 ppm) for chinook salmon (Katz 1961). Only 13 percent of carp embryos exposed to one part per million survived to hatch; 0.1 ppm did not increase embryonic mortality, but resulted in 100 percent mortality of all fry within 48 hours after eclosion (Malone and Blaylock 1970). The toxicity of azinphosmethyl to bluegills and to rainbow trout increased with increasing temperatures (Macek et al. 1969). At levels used for controlling mosquito larvae, azinphosmethyl was reportedly not toxic to the mosquitofish (*Gambusia*) or to pollywogs (Mulla et al. 1963).

Birds--The acute oral LD_{50} of azinphosmethyl was 125 to 150 mg/kg in mallards (Keith and Mulla 1966) and 277.3 mg/kg in 10 to 12 day old chickens (Sherman et al. 1967). The LC_{50} of azinphosmethyl to baby chicks was 1197 ppm, but levels above 300 ppm decreased growth rates within one week (Steve et al. 1961). Injection of one mg azinphosmethyl into chicken eggs was teratogenic (Upshall et al. 1968). Chickens fed 40 mg/kg azinphosmethyl developed leg weakness (Gaines 1969).

Mammals--The acute oral LD_{50} of azinphosmethyl in rats is 15 mg/kg (Eto 1974). No data were available on the carcinogenicity, mutagenicity, teratogenicity, or other reproductive effects of azinphosmethyl.

Conclusions-

Despite its apparently rapid dissipation in soil, and its consequently minimal leaching, the feasibility of disposing of azinphosmethyl in soil is not demonstrated by the data. Staiff and co-workers (1975) have indicated a tendency for higher levels of soil contamination to resist degradation; no contrary data are available. There is plentiful evidence, however, that temperature and moisture exert a strong effect on the rate of azinphosmethyl degradation. Therefore, the least objectionable site for soil disposal of azinphosmethyl wastes would be a moist, semitropical soil. Since wettable powders appear to degrade more quickly, but leach more readily than emulsifiable concentrates, no conclusions concerning relative risks of different formulations can be drawn.

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CARBAMATE INSECTICIDES

Carbaryl

Carbaryl is the common name for 1-naphthyl *N*-methyl carbamate, introduced by the Union Carbide Corporation in 1956 as Sevin. A contact insecticide with slight systemic properties, carbaryl is made by reaction of 1-naphthol and methyl isocyanate or by reacting 1-naphthol with phosgene and then methylamine. It is a white crystalline solid with a melting point of 142°C, a vapor pressure of 0.005 mm mercury at 26°C, and a water solubility of less than 0.1 percent (1,000 ppm) at room temperature. It is soluble in most polar organic solvents such as dimethyl sulfoxide.

Degradation--

Biological--The degradation of carbaryl by microorganisms is summarized in Tables 81 and 82 and the pathways are shown in Figure 17. The degradation to 1-naphthol proceeds by hydrolysis, hydroxylation and possibly also by conjugation (Mehendale and Dorough 1972). Bollag and Liu (1971) noted that 1-naphthol is both more toxic and more persistent than carbaryl itself. Degradation of carbaryl to CO_2 and coumarin has been reported for *Pseudomonas* (Kazano et al. 1972). Bollag and Liu (1971) reported that *Fusarium solani* degraded 1-naphthol more readily than carbaryl, and that filamentous fungi were more effective than bacteria. Of 11 microorganisms tested, three were completely unable to degrade carbaryl (Sikka et al. 1975). *Trichoderma viride* decomposed 11.3 percent of ^3H-carbaryl, but only 2.1 percent of the label was water-soluble (Matsumura and Boush 1968). *Aspergillus terreus* degraded carbaryl when it was present at 50 to 100 ppm, but was completely inhibited by 200 to 500 ppm. The pyrethrin-synergizing methylenedioxyphenyl compounds, Sesamex and Sesamol, also decreased the rate of car-

Table 81. DEGRADATION OF CARBARYL BY BACTERIA

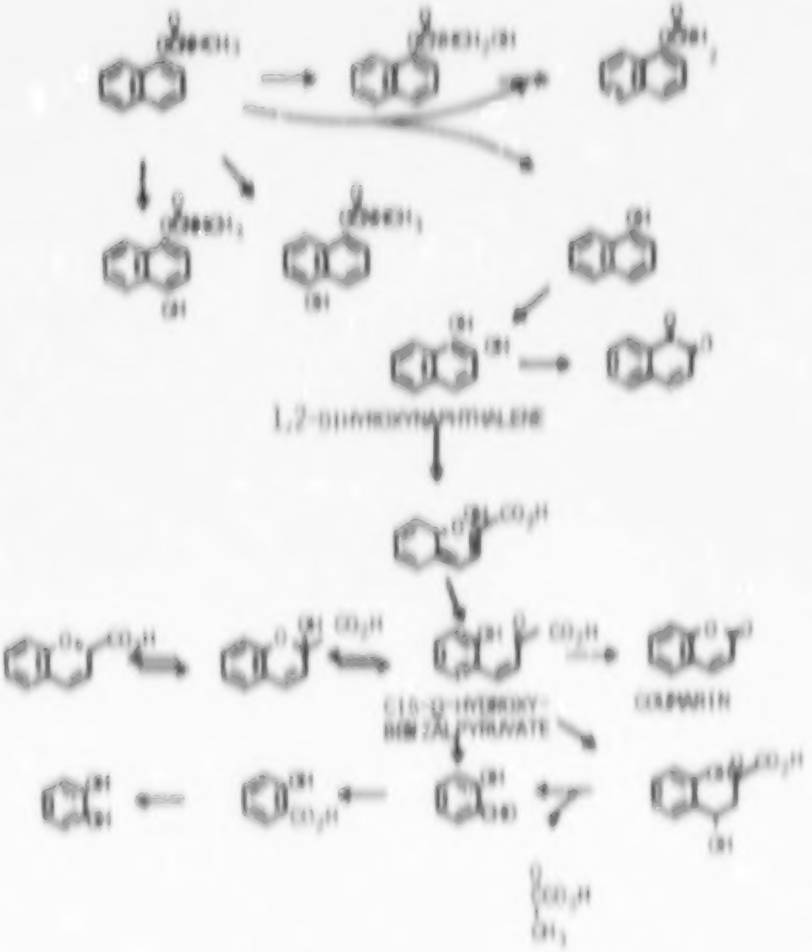
Organism	Conditions	Products	Reference
Bacteria, soil		4, unidentified	Tewfik and Hamdi 1970
" S-1		4, unidentified	Lohoda 1970
Gram$^-$ coccus C	silt loam culture		Bollag and Liu 1971
Gram$^+$ rod B	silt loam culture	1-naphthol	Ibid.
Achromobacter	Sevin as sole C-source	1-naphthol, hydroquinone, catechol, pyruvate	Sud et al. 1972
Bacillus cereus	cultures	unidentified	Singh 1974
Pseudomonas melophthora	^{14}C-carbaryl	48.1% solvent-soluble 6.4% water-soluble	Boush and Matsumura 1967
Pseudomonas sp.	culture	CO$_2$, coumarin, 1-naphthol	Kazano et al. 1972

Table 81. DEGRADATION OF CARBARYL BY FUNGI

Organism	Conditions	Products	Reference
Aspergillus terreus	synergistic; culture	1-naphthyl carbamate	Bollag and Liu 1974
	50-100 ppm carbaryl	1-naphthyl N-hydroxymethyl carbamate	Liu and Bollag 1971b
Trichoderma viride	^{14}C-carbaryl	11.3% degradation	Matsumura and Boush 1968
Zalerion maritimum	marine microorganisms		Sikka et al. 1975
Candida parapsilosis[a]	" "		Ibid.
Rhodotorula glutinis[a]	" "		Ibid.
Trichosporon fermentans[a]	" "		Ibid.
Aspergillus fumigatus	" "		Ibid.
Culcitalna achraspora[b]	" "		Ibid.
Halosphaeria mediosetigera[b]	" "		Ibid.
Humicola alopallonella	" "		Ibid.
Gliocladium roseum		1-naphthyl(hydroxymethyl) carbamate 4-hydroxy-1-naphthyl methylcarbamate 5-hydroxy-1-naphthyl methylcarbamate	Liu and Bollag 1971a

[a] Also degrade 1-naphthol

[b] Best with carbaryl


1,2-DIHYDROXYNAPHTHALENE
CIS-O-HYDROXY-BENZALPYRUVATE
COUMARIN

MICROBIAL METABOLISM
OF CARBARYL
(AFTER KAUFMAN, 1974)

Figure 17

baryl degradation by *Aspergillus* (Bollag and Liu 1974). An *Achromobacter* species converted carbaryl to 1-naphthol, hydroquinone and catechol pyruvate in the absence of other sources of carbon (Sud et al. 1972). *Pseudomonas melophthora*, a symbiote of the apple maggot, converted 6.4 percent of ^{3}H-carbaryl to water-soluble metabolites and 45.5 percent to solvent-soluble metabolites (Roush and Matsumura 1967) while another symbiote, *Bacillus cereus*, reportedly also degraded carbaryl (Singh 1974).

When ^{14}C-1-naphthol was added to cultures of bacteria in river water, 44 percent of the 1-naphthol was converted to ^{14}C-carbon dioxide within 60 hours. Seventeen percent of the radioactivity remained in the growth medium, and 22 percent was associated with the bacteria. The major residue was 4-hydroxy-1-tetralone, and bacterial growth was completely inhibited by more than 100 ppm of 1-naphthol (Bollag et al. 1975).

Metabolism of carbaryl in plants may consist of little more than inactivation; alternatively, oxidation or hydroxylation of the *N*-methyl group may be followed by ring degradation (Kuhr 1968).

In fish (*Cyprinus carpio*), Ishii and Hashimoto (1970) reported complete degradation of carbaryl, with no 1-naphthol detectable after 24 hours. In cows, 1-naphthol and conjugated metabolites of carbaryl were excreted in the urine and feces, but not in milk, when cows were fed 450 ppm of technical carbaryl for fourteen days. Urinary excretion ended within four days of the last exposure to carbaryl, and even ingestion of 12 ppm of 1-naphthol did not result in tainted milk (Whitehurst et al. 1963). Rat intestines converted carbaryl to napthyl glucuronide *in vitro* (Pekas 1971).

In human embryonic lung cells in culture, carbaryl was almost completely metabolized within 72 hours. The major metabolites were 1-naphthol, 4-hydroxycarbaryl, 5-hydroxycarbaryl, and 5,6-dihydroxycarbaryl. Unknown compounds and conjugates were also produced (Lin et al. 1975).

In mice, both 4-hydroxycarbaryl and 5-hydroxycarbaryl are rapidly conjugated to form *beta*-D-glucosides, which are significantly less toxic to the animals (Cardona and Dorough 1975).

Chemical and physical--Treatment with liquid ammonia and metallic sodium or lithium destroyed 92.8 and 93.5 percent of analytical grade carbaryl, respectively, but the decomposition products were not identified (Kennedy et al. 1972a). Treatment with 16N nitric acid resulted in formation of nitrobenzene; 15N ammonium hydroxide only resulted in conversion to 1-naphthol (Kennedy et al. 1972b). In soil, nonbiological hydrolysis of carbaryl to 1-naphthol has been shown to occur (Kazano et al. 1972).

Thermal degradation of carbaryl in commercial formulation was 88.7 percent effective at 600°C, and 89.5 percent effective in a dry combustion furnace at 1,000°C (Kennedy et al. 1969). Volatile products of carbaryl combustion at 900°C included carbon monoxide, carbon dioxide, hydrochloric acid, ammonia, and oxygen, as well as unidentified products (Kennedy et al. 1972a, 1972b).

Photolytic--Ultraviolet light decomposed carbaryl primarily by cleavage of the ester bond (Aly and El-Dib 1971, Fedorova and Karchik 1970). Further photolytic studies demonstrated that some degradation products were also cholinesterase inhibitors and that the nature of the degradation depended on formulation (Crosby et al. 1965). Moisture (Fedorova and Karchik 1970) and high pH (Aly and El-Dib 1971) accelerated photolysis of carbaryl.

Transport:

Within soil--Carbaryl was less readily adsorbed to pond sediments and watershed soils than either malathion or phorate (Meyers et al. 1970). After 1,000 liters/ha of three percent Sevin were applied to soil, 51.5 to 58.1 percent of the original level of carbaryl was detected between zero and five centimeters after five days; residues were detected at depths of 50 to 60 centimeters for 15 to 20 days (Halbandyan 1974). Adsorption of carbaryl to soil organic matter surfaces is probably physical rather than chemical, depends on the saturating cation and increases with increasing temperature between 5° and 40°C (Leenheer and Ahlrichs 1971).

Between soil and water--No data were available on the transport of carbaryl or its major metabolite, 1-naphthol, through or with soil into water, or from water into sediments.

Into air--Loss of carbaryl from petri dishes after 12 days exposure to a constant air flow of ten cubic feet per hour was 1.6 mg at a relative humidity of three to 13 percent, and 6.2 mg at a relative humidity of 75 to 85 percent, but the amounts originally exposed were not given (Lyon and Davidson 1965).

Into organisms--Carrots contained less carbaryl than either parathion or diazinon after fields were treated with two or three times the normal agricultural levels of each chemical (Stobwasser 1963).

In the subarctic, residues of carbaryl one month after application of five kg/ha (2.2 ppm) were 0.1 ppm in soil, 0.5 ppm in lichens, 0.6 ppm in arctic birds, 1.6 ppm in lemming livers, 1.5 ppm in woodcock livers, and ten ppm in the testes of small mammals (Shilova et al. 1973). Kurtz and Studholme (1974), however, found little uptake of carbaryl in songbirds after forests were sprayed for gypsy moths, and no carbaryl residues were present in cattle after seven days, although milk excretion persisted for over 60 hours (Dorough 1967). Organ retention of carbaryl in mice lasted two days after doses of 50 mg/kg; in rabbits, six days after doses of 400 mg/kg; and in chickens, five days after 1,500 mg/kg (Ryamushkin and Yakub 1971).

Persistence-

Carbaryl applied as a termiticide retained 60 to 100 percent of its effectiveness for four years (Beal and Smith 1972). Ivanova and

Moloshanova (1974) postulated an exponential decrease in soil levels of carbaryl with time; empirically, the rate of loss was:

$$1/k = 16.9\ a + 15.3\ pH - 46.24$$

where a is equal to the humus content of the soil.

In river water exposed to natural and artificial light in jars, 95 percent of ten g/liter (10,000 ppm) of carbaryl decomposed within one week, and no carbaryl was detected after two weeks (Eichelberger and Lichtenberg 1971). In laboratory aquaria, the "half-life" of carbaryl in seawater without mud was 38 days at $8^{\circ}C$, but at $20^{\circ}C$, 43 percent was converted to 1-naphthol in 17 days. When mud was included in the aquaria, less than ten percent of the carbaryl was converted to 1-naphthol after ten days. In a natural mud flat, carbaryl persisted for 42 days, 1-naphthol for only one day (Karinen et al. 1967).

Soil residues one month after five kg/ha were applied to soil in the subarctic were 0.1 mg/kg (0.1 ppm) (Shtilova et al. 1973). Kazano et al. (1972) noted that 1-naphthol was chemically bonded to the humus in soil and was stable to alkali.

Effect on Non-Target Species:

Microorganisms--In the laboratory, while 20 ug/ml (20 ppm) inhibited growth of *Fusarium oxysporum*, the effects could be prevented by yeast extracts, asparagine, ammonium nitrate, or ammonium sulfamate, but not by vitamins. None of the 17 fungi tested was able to grow with carbaryl as the sole source of carbon (Cowley and Lichtenstein 1970). Jakubowska and Novak (1973) reported significant inhibition of fungal growth by carbaryl, but in another study carbaryl stimulated fungal growth (Askerov 1969). Algae and bacteria varied in their response, as summarized in Tables 83 and 84. Carbaryl in sandy soil produced a greater effect on microorganisms than in either chernozem or peat (Brantsevich et al. 1973).

Invertebrates--The acute LD_{50} of carbaryl to honeybees was 0.02 percent, or 200 ppm, under laboratory conditions. Under the experimental

Table 81. EFFECTS OF CARBARYL ON SOIL MICROORGANISMS

Parameter	Effect	Level Original	 PPM	observed	Conditions	Reference
Bacterial growth	–	3 kg/ha	1.4	1085	peat, sand, chernozem	Bruestsevich *et al.* 1973
Azotobacter	–	20,50 ppm	20,50	1	normal after 1st day	Gaur and Misra 1970
Bacillus subtilis	–	0.07%	700			DeGiovanni-Donnelly *et al.* 1968
Bacillus thuringiensis	–				culture	Chen *et al.* 1974
Soil bacteria	0	1–1.2 kg/ha	<0.5	10	single application	Ankarov 1969
Soil bacteria	+	1–1.2 kg/ha	<0.5	10	repeated applications	Ibid.
Actinomycetes	0	1–1.2 kg/ha	<0.5	10	single application	Ibid.
	–					Jakubowska and Nowak 1973
Fungi	0	1–1.2 kg/ha	<0.5		single application	Ibid.
	+	1–1.2 kg/ha	<0.5		repeated applications	Ibid.
	–	20,50 ppm	20,50			Gaur and Misra 1970
	0	1 ppm	1			Ibid.

+ = stimulation
0 = no effect
– = inhibition

Table 84. EFFECTS OF CARBARYL ON AQUATIC MICROORGANISMS

Parameter	Effect	Level Original	 PPM	# days observed	Conditions	Reference
Scenedesmus quadricauda	+	0.1-1.0 ppm	0.1-1.0		static culture	Stadnyk et al. 1971
Chlorella pyrenoidosa	–	<0.10	<0.1			Christie 1969
Protococcus sp.	–	0.1-100	0.1-100	10	seawater culture	Ukeles 1962
Chlorella sp.	–	0.1-100	0.1-100	10	seawater culture	Ibid.
Dunaliella euchlora	–	0.1-100	0.1-100	10	seawater culture	Ibid.
Phaeodactylum tricornutum	–	0.1-100	0.1-100	10	seawater culture	Ibid.
Monochrysis lutheri	–	0.1-100	0.1-100	10	seawater culture	Ibid.
Fusarium oxysporum	–	20 μg/ml	20	1	laboratory	Cowley and Lichtenstein 1970

+ = stimulation
0 = no effect
– = inhibition

conditions used, carbaryl was less toxic to bees than parathion, dieldrin, or azinphosmethyl, but more toxic than phorate or DDT (Johansen 1961). In pasture, carbaryl reduced the biomass of earthworms by as much as 60 percent, and their numbers by as much as 69 percent (Thompson 1970); straw decomposition was delayed by 6.5 to 17.6 percent because of carbaryl toxicity to the earthworm *Allolophora caliginosa* (Atlavinyte et al. 1974).

At 2.3 and 4.6 kg/ha on intertidal mud flats, carbaryl decreased the numbers of juvenile clams (*Tresus capax*, *Macoma nasuta*, *Callianassa californiensis*), but did not appear to reduce the numbers of polychaete or nemertean worms (Armstrong and Millemann 1974). The harvest of Louisiana red crawfish, *Procambarus clarkii*, was unaffected by carbaryl at agricultural levels (Hendrick et al. 1966).

In an aquatic ecosystem (Kanazawa et al. 1975), carbaryl was characterized as relatively persistent when it was applied to air-dried soil, after which water and aquatic organisms were added. Most of the carbaryl (68 percent) remained in the soil; 0.11 to 1.19 percent was taken up by the catfish, crawfish, daphnids, algae, and duckweed. No toxicity was associated with the soil-bound carbaryl, 45 percent of which was not extractable by solvents or methanol (Kanazawa et al. 1975).

In an aquatic-terrestrial model ecosystem (Metcalf et al. 1971), no carbaryl residues were found in any of the organisms, and the water contained only degradation products. Sanborn (1974) and Metcalf and Sanborn (1975) concluded that carbaryl would not accumulate in aquatic food chains.

Plants--Carbaryl increased the herbicidal persistence of CIPC (isopropyl m-chlorocarbanilate) regardless of soil pH or soil type (Kaufman et al. 1972). Propanil inhibited interconversion of carbaryl metabolites on leaves (Chang et al. 1971b), and carbaryl inhibited the metabolism of propanil in leaf tissue (Chang et al. 1971a) and in soil (Kaufman et al. 1971). Chlorpropham and malathion stimulated the degradation of carbaryl on leaves, but linuron inhibited it (Chang et al. 1971b).

On grain sorghum, agricultural levels of carbaryl were not phytotoxic (Meisch et al. 1970). Soaking cotton seeds (*Gossypium barbadense*) in carbaryl for one to three days decreased seed germination, but weekly sprays of a 50 percent saturated solution increased both abscission and cotton yields per plant (Hammouda et al. 1966).

At 1,500 ppm, carbaryl decreased the germination of barley (*Hordeum vulgare*) by 52 percent, and induced chromosome aberrations in root tip cells (Wuu and Grant 1966). Chromosome aberrations were induced in beans (*Vicia faba*) by spraying plants with carbaryl daily for eight days. Pollen sterility was not induced, but the chromosome damage was dose-dependent (Amer and Farah 1968).

Fish--The 24-hour LC_{50} of carbaryl to fish ranged from 1.75 ppm for longnose killifish (*Fundulus similis*) to 6.7 ppm for three-spine sticklebacks (*Gasterosteus aculeatus*), while the 96-hour LC_{50} ranged from 0.76 ppm in coho salmon (*Oncorhynchus kisutch*) to 20 ppm in black bullheads, (*Ictalurus melas*). Some data suggest that sub-clinical levels of carbaryl lowered the natural resistance of fish to parasites (Pimentel 1971).

Birds--The acute oral LD_{50} of carbaryl in Japanese quail (*Coturnix coturnix japonica*) was 2,290 mg/kg and in mallards (*Anas platyrhynchos*) more than 2,179 mg/kg (Tucker and Crabtree 1970). In 39 day old Japanese quail, liver levels of vitamin A were decreased in females but not in males (Cecil et al. 1974). The acute oral LD_{50} in male chicks was 197 mg/kg, with 95 percent confidence limits ranging from 156 mg/kg to 250 mg/kg (Sherman and Ross 1961), but Zhavoronkov et al. (1973) claimed that after feeding one percent of the LD_{50} to hens for ten days, development of their chicks was impaired.

When 1.45 mg/embryo of carbaryl was injected into the allantoic cavity, 50 percent of chick embryos died, but the survivors exhibited no histopathologic changes, even when 0.75 mg/embryo were injected ([illegible] et al. 1973). Olefir and Vinogradova (1968) reported numerous malformations in chick embryos after 0.064 mg/kg carbaryl were injected into the yolk sac. Dunachie and Fletcher (1969) observed malformations, mainly of the feathers, in chicks treated with [illegible] ppm carbaryl injected into the yolk.

Mammals--The acute oral LD_{50} of carbaryl in rats is between 500 mg/kg (Jones et al. 1968) and 850 mg/kg (Metcalf et al. 1962). Yakim (1967) cited an acute oral LD_{50} of 721 mg/kg for rats and 150 mg/kg for cats. Since rats survived daily injection of five percent of the LD_{50} for at least six months, Yakim (1967) considered carbaryl no more than mildly cumulative. Pretreatment with DDT decreased the toxicity of carbaryl in mice (Meksongsee et al. 1967).

Numerous reports suggest that carbaryl exerts a considerable effect on cellular enzymes (Khalkina 1970, Orlova 1970, Kagan et al. 1970, Kuz'minskaya and Yakushko 1970, Kuz'minskaya 1971), on the immune response (Olefir 1971), and causes cardiovascular disturbances (Orzel and Weiss 1966, Kagan et al. 1970, 1973, 1975; Lukaneva and Rodionov 1973). In tissue culture, carbaryl was more toxic than its metabolite 1-naphthol (Litterst and Lichtenstein 1971). Lower concentrations of carbaryl stimulated the growth of HeLa cells, but at higher concentrations growth was inhibited (Blevins and Dunn 1975). Ten percent of the LD_{50}, given daily for thirty days, altered the exocrine activity of the pancreas (Urbanowicz et al. 1973).

Reproductive disturbances in rats have been reported at chronic treatment with no more than 50 mg/kg/day (Shtenberg et al. 1970, Shtenberg and Ozhovan 1971, Przezdziecki et al. 1974, Trifonova et al. 1970). Embryotoxic effects were also reported by several authors (Shtenberg et al. 1973, Tam-Lutz et al. 1974, Torchinskii 1974). In a three-generation study, chronic feeding of 2,000 ppm decreased the reproductive fitness of both rats and gerbils (Collins et al. 1971). In mice, no significant effects on reproduction were observed when ten mg/kg of carbaryl were fed for three generations, and even 500 mg/kg produced no significant effects except an increase in pre-weaning mortality. Administration of 100 mg/kg by intubation before mating did, however, reduce fertility (Weil et al. 1972).

Carbaryl was not teratogenic in guinea pigs at 300 mg/kg, in hamsters at 250 mg/kg, or in rabbits at 200 mg/kg (Robens 1969). Carbaryl is not teratogenic in rats (Shtenberg and Torchinskii 1972, Weil et al. 1973). In beagle dogs, Smalley and co-workers (1968) observed malformations at carbaryl levels of 50 mg/kg, but no dose-response relationship was evident. There was no evidence of chromosome damage in mice injected intraperitoneally with 20 mg/kg of carbaryl (Jordan et al. 1975) and no increase in resistance to carbaryl after 12 to 14 generations of selection (Guthrie et al. 1971).

Makovskaya and co-workers (1965) reported fatty necrotic areas in the livers and spleens of mice treated with 60 mg/kg carbaryl for six months; no tumors were found even after 20 months. Zabezhinski (1970) claimed that Seto-Sevin caused "cancerous tumors" when given orally or intravenously, but no dosage level was reported. Carbaryl can also serve as a precursor to the potent bacturial mutagen, nitrosocarbaryl (Elespuru et al. 1974, Siebert and Eisenbrand 1974) which could plausibly be formed within the human digestive system (Uchiyama et al. 1975).

Mild, permanent, and increasing functional deviation was observed in the nervous system of maze-trained rats treated with subacute levels of carbaryl (Desi et al. 1974).

Conclusions:

Inasmuch as both carbaryl and its major metabolite 1-naphthol are readily decomposed by microorganisms, soil disposal of carbaryl is feasible.

Metalkamate

Metalkamate, more commonly referred to as Bux, is a mixture of three parts *m*-(1-methylbutyl)-phenyl *N*-methylcarbamate and one part of *m*-(1-ethylpropyl)-phenyl *N*-methylcarbamate, introduced by Chevron Chemical Company in 1966 under the trade names of Ortho 5353 and Bux. Metalkamate is a colorless solid with a melting point of 45 to 50°C. It is soluble in water to less than 50 ppm, but readily soluble in xylene and methanol. Bux is formulated as ten percent clay granules, and is used primarily against corn rootworm larvae (Tucker 1973).

Degradation:

Tucker and Pack (1972) analyzed the degradation of metalkamate in soil under laboratory conditions and concluded that microbial degradation predominated, since degradation was more rapid in unsterilized than in sterile soil. In unsterilized soil, 50 percent of the metalkamate was degraded within one week and in sterilized soil, five to fifteen percent was degraded in the same time with $^{14}CO_2$ evolution from ^{14}C-carbonyl-labeled metalkamate. In additional studies on the major component, *m*-(1-methylbutyl)-phenyl *N*-methylcarbamate, the major metabolite was *m*-(1-hydroxy-1-methylbutyl)-phenyl *N*-methylcarbamate.

Metalkamate is stable in neutral or acidic media, but under alkaline conditions it is hydrolyzed to the respective phenols, carbon dioxide, and methylamine (Tucker 1973). No data were available on the photolysis or physical degradation of metalkamate.

Transport:

No data were available on the movement of metalkamate in soil, between soil and water, or on losses due to volatilization.

In an aquatic-terrestrial model ecosystem, metalkamate did not show any tendency to accumulate in the higher members of the trophic web.

The alga and *Elodea* contained residues of 0.98 ppm and 0.26 ppm, respectively, of the parent compound; and the crab, which died seven days after the addition of metalkamate, contained 0.05 ppm. The other organisms contained no detectible amounts of ^{14}C. Yu et al. (1974) and Metcalf and Sanborn (1975) concluded that metalkamate would not lead to environmental problems of accumulations in the food chain.

Persistence:

Harris (1973) considered metalkamate to be a moderately persistent soil insecticide which lost its insecticidal activity within 16 weeks. Metalkamate was less toxic to crickets when soil-applied than as a direct contact insecticide, but the inactivation was only moderate in moist sand. In muck, activity against crickets was reduced by a factor of fifty in comparison to direct toxicity (Harris, 1973). Degradation of metalkamate in solution was 50 percent complete in silt or silt loam soil in one week under laboratory conditions. The authors concluded that slower degradation would occur under field conditions since Bux is applied as granules (Tucker and Pack 1972).

Effects on Non-Target Species:

No data were available on the effects of metalkamate on soil processes or soil microorganisms. Apple (1971) considered Bux sufficiently phytotoxic that in-row application at the rate of one pound per acre might injure corn with damage depending on climatic conditions in the days following planting. Bux applied at one pound per acre reduced the numbers of earthworms in pasture severely (Thompson 1970), but recovery occurred within one year (Thompson and Sans 1974). The acute oral LD_{50} of Bux is 87 mg/kg in rats, and the acute dermal LD_{50} is 500 mg/kg (Anonymous 1974).

Conclusions:

Metalkamate is toxic to earthworms in pasture (Thompson 1970, Thompson and Sans 1974), is phytotoxic to corn at approximately 2 ppm, or 1 lb/A

(Apple 1971), and appears to be highly toxic to crabs (Metcalf and Sanborn 1975).

In the absence of data on its transport within soil and from soil to water, soil disposal of metalkamate wastes is not recommended. Prior treatment by chemical hydrolysis to the phenols before soil disposal might greatly enhance the degradation, but the consequences are merely speculative.

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SECTION V
FUNGICIDES AND FUMIGANTS

Captan

Captan is the common name for 3a,4,7,7a-tetrahydro-2-(trichloromethyl) thio-1H-isoindole, a fungicide used mainly for foliage protection which was introduced by Standard Oil in 1949 under the name Orthocide. Captan forms white crystals with a melting point of 178°C and a vapor pressure of less than 0.01 mm at 25°C. It is insoluble in petroleum oils, and its water solubility at room temperature is less than 0.5 ppm. It is soluble in xylene, chloroform, acetone and isopropanol, and stable except under alkaline conditions.

Captan is produced by the reaction of trichloromethylsulphenyl chloride on tetrahydrophthalimide. The technical product is 93 to 95 percent pure captan and forms an amorphous yellow solid with a pungent odor. The melting point of technical captan is between 160° and 170°C. It is formulated as 50 percent or 83 percent wettable powders, 5 percent dust, or as a 75 percent dust for seed treatment.

Degradation-

Biological--When 840 million *Neurospora crassa* spores were incubated with 375 ug captan, degradation of captan to carbonyl chloride occurred in 20 minutes, with no formation of carbon disulfide (Somers *et al.* 1967, Richmond and Pickard 1967). Cell thiols were implicated in the degradation and most of the ^{35}S was eventually bound to oxidized glutathione and its derivatives (Richmond and Somers 1968). Captan lost 50 percent of its bioactivity against *Rhizoctonia solani* after three weeks' incubation in humus-sandy forest soil (Kluge 1969a).

Rats fed ^{14}C-labeled captan excreted 51.8 percent of the ^{14}C in their urine as thiazolidine-2-thione-4-carboxylic acid, a salt of dithiobis (methane sulfonic acid) and the disulfide monoxide of dithiobis(methane sulfonic acid). Radioactive CO_2 was exhaled, accounting for almost 23 percent of the label, and 15.9 percent of the label was eliminated in the feces. Less than one percent of the label remained in the tissues (DeBaun *et al*. 1974). Gastro-intestinal degradation was highly significant, since in rats which were injected intraperitoneally with captan, 50 percent of the radioactive label was excreted in nine days, but administered orally the label from captan was 50 percent excreted within two hours (DeBaun *et al*. 1974).

When ^{35}S-labeled captan was fed to rats, 90 percent of the label was eliminated in 24 hours (Seidler *et al*. 1971).

Chemical and physical--Captan decomposes between 200°C and 245°C leading to a 52 percent weight loss and formation of hexachlorodimethyl disulfide and tetrahydrophthalimide (Pfeifer and Pfeifer 1970). Captan degradation in soil was not affected by changes in pH between 3.6 and 7.6 (Kluge 1969b).

Transport--

When the mobility of several fungicides was examined in soil columns with several types of soil, all were more mobile in sandy loam than in loam, and in dry than wet soil. Peat moss greatly inhibited the mobility of all compounds. In general, suspended compounds were less mobile than dissolved compounds. Captan as particles of 14.5 µ was less mobile than when particles of 1.05 µ were used (Munnecke 1961).

Persistence--

Captan was found to be degraded rapidly in most soils. Bioassay with *Myrothecium verrucaria* indicated a 50 percent loss of activity after seven days in sandy soil, three to four days in moist soil, and less than one day in compost soil. Under conditions of high local concentrations, much longer persistence was noted (Griffith and Matthews 1969). Agnihotri (1970, 1971) and Burchfield (1959) also found that captan was persistent for less than one week in moist soil.

In sharp contrast, Munnecke (1958) found persistence of diffusible activity for 65 days after captan was applied to a mix of sand and peat while under the same conditions nabam was inactivated within hours. If the mix was first sterilized, captan persisted for up to 150 days. Agnihotri (1970), observing that Munnecke's data were atypical, suggested that the acidity of the peat moss was unfavorable for growth of captan-degrading bacteria. The amount of organic matter, rather than clay, in the soil determines the rate of captan release (Persheim and Linn 1968). Fifty percent of the applied captan persisted for less than one hour in water at 25^{0}C, but for seven hours at 12^{0}C (Hermanutz et al. 1973).

These data suggest that biodegradation of captan is rather rapid, which explains the nonpersistence of captan under field conditions. No data on the degradation products of captan or their persistence in soil were available.

Effects on Soil Microorganisms-

As would be expected of a fungicide, captan sharply inhibited nontarget fungal growth in many studies (Wainwright and Pugh 1974, Naumann 1970, Domsch 1959) but after 28 days fungal populations had increased above pretreatment levels even if twice the usual agricultural levels of captan were used. *Gliocladium*, *Penicillium*, and *Trichoderma* predominated (Wainwright and Pugh 1974). A decrease in fungal diversity was observed by Naumann (1970). When captan was applied to forest soil at 62.5, 125, and 250 ppm, three pathogenic fungi were destroyed (*Rhizoctonia*, *Pythium*), three saprophytic fungi were stimulated (*Penicillium*, *Trichoderma*, *Fusarium*) and actinomycetes and bacteria were stimulated for up to five weeks (Agnihotri 1970, 1971). In laboratory cultures, agricultural levels of captan inhibited four microfungi (*Hansenula saturnus*, *Mucor hiemalis*, *Pennicillium stipitatum* and *Trichoderma viride*) from cattail marsh, but in the field no effects on these fungi were observed (Tews 1971).

Roeder et al. (1969) found captan almost as toxic to some algae as to *Neurospora crassa*: 37 of 40 *Chlorella* species were strongly inhibited by as little as five mg/liter (five ppm) captan in shake solution whereas *Scenedesmus* species were relatively resistant even at 50 mg/liter (50 ppm) of captan. With respect to nontarget microorganisms and soil processes, captan generally stimulated ammonification and inhibited nitrification, while bacterial growth was sometimes stimulated and sometimes inhibited. These data are summarized in Tables 85 and 86.

In addition to the considerable direct toxicity which it exerts on some microorganisms, captan induces DNA base changes (Kada et al. 1974). It is mutagenic in the bacteria *Escherichia coli* (Legator et al. 1969, Clark 1971, Bridges et al. 1972, 1973) and *Salmonella* (Seiler 1973), in the fungus *Alternaria mali* (Slifkin 1973), and induces mitotic gene conversion in the yeast *Saccharomyces cerevisiae* (Siebert et al. 1970). In the host-mediated assay in mice, captan was mutagenic in two species of *Salmonella* (Buselmaier et al. 1972).

In summary, it is apparent that captan strongly affects soil microorganisms and soil processes and must be expected to alter the soil complex profoundly whenever it is applied to soil, even if the precise changes cannot be predicted.

Effect on Non-Target Species-

Invertebrates--Captan is essentially nontoxic to insects under ordinary usage (Pimentel 1971, Beran and Neururer 1956).

Plants--Captan was reported to stimulate plant growth (Kittleson 1963), but 1,000 ppm inhibited root growth of corn seedlings (Dugger et al. 1958). Levels of 100 and 200 ppm of a 50 percent wettable powder decreased corn growth but stimulated growth of bean plants in soil consisting of silty clay loam, peat and fine sand in a 3:1:1 ratio (Cole et al. 1968).

Fish--Ninety percent of zebrafish larvae exposed to one ppm of captan died within 90 minutes (Abedi and McKinley 1967). Lethal concentrations were reported to be 29 ppm for brook trout (*Salvelinus fontinalis*), 64

Table 85. EFFECTS OF CAPTAN ON SOIL PROCESSES

Parameter	Effect	Dose Original	PPM	# days Observed	Soil or conditions	Reference
CO_2 production	-	≤250 ppm	≤250	35	forest soil	Agnihotri 1970, 1971
Ammonification	0	≤250 ppm	≤250	35	forest soil	Ibid.
	+	ag levels		>84	several soils	Wainwright and Pugh 1974
	0	5 ug/gram	5		+ $(NH_4)_2SO_4$	Pugh 1973
	+	<250 ug/gram	250		+ $(NH_4)_2SO_4$	Ibid.
Nitrification	0					Capriotti and Martini 1959
	-			14	forest soil	Agnihotri 1970, 1971
	-	ag levels		>84	several soils	Wainwright and Pugh 1974
	+	5 ug/gram	5		+ $(NH_4)_2SO_4$	Pugh 1973
	-	250 ug/gram	250		+ $(NH_4)_2SO_4$	Ibid.
	-	2%	20,000		agar	Picci 1956
	0	0.1%	1,000		agar	Ibid.

+ = stimulation
0 = no effect
- = inhibition

Table 86. EFFECTS OF CAPTAN ON SOIL MICROORGANISMS OTHER THAN FUNGI

Parameter	Effect	Dose Original	Dose PPM	# days Observed	Soil or conditions	Reference
Bacteria	+			7		Agnihotri 1970, 1971
	0			35		Ibid.
	+	2x ag levels		>28		Wainwright and Pugh 1974
	-	0.1%	1,000		agar	Picci 1956
	0					Domsch 1959
Azotobacter	-	≥0.1%	≥1,000		agar	Picci 1956
A. chroococcum	-	250 ppm	250			Naumann 1970
Cellulolytes	-					Capriotti and Martini 1959
Actinomycetes	-	≤250 ppm	250	<35		Agnihotri 1970, 1971
	0	≤250 ppm	250	35		Ibid.
	-					Domsch 1959
	0	250 ppm	250			Naumann 1970
Algae	-					Domsch 1959
Chlorella sp.	-	250 mg/ml			shake culture	Sender *et al.* 1969
Scenedesmus		250 mg/ml			shake culture	Ibid.

+ = stimulation
0 = no effect
- = inhibition

ppm for fathead minnows (*Pimephales promelas*), and 72 ppm for bluegills (*Lepomis macrochirus*) and the breakdown products of captan were not toxic (Hermanutz et al. 1973).

Birds--Captan is relatively nontoxic in birds with a five-day LC_{50} of 2,000 ppm in bobwhite and over 5,000 ppm in mallards, pheasants, and coturnix quail (Pimentel 1971). If captan is injected into hens' eggs it causes leg and wing malformations as well as significant mortality (Verrett et al. 1969).

Mammals--The acute oral LD_{50} of captan is more than 8,000 mg/kg in rats (Jones et al. 1968), but 250 mg/kg caused poisoning in sheep (Palmer and Radeleff 1964). Chronic feeding of 0.05 to 0.4 ppm for six months did not affect two cows or five swine so fed (Johnson 1954), while 140 days feeding with corn contaminated by 67 g/bushel apparently increased weight gain in steers (Dowe et al. 1957). In contrast, Grabarska (1972) reported serious dystrophic changes in many organs when rabbits were given 500 mg/kg for 14 days and similar damage was reported by Vashakidze et al. (1973) when 265 mg/kg were given to rats for two months, or 20 to 55 mg/kg were given for twelve months. Treatment with 100 mg/kg/day of captan decreased the level of hepatic respiratory enzymes in guinea pigs (Krolikowska-Prasal 1973).

Since captan was observed to denature calf thymus *in vitro* and cause alkylations *in vivo* in mice it was considered carcinogenic by Andersen and Rosenkranz (1974). The teratogenicity of captan is theoretically attractive because it shares the phthalimide moiety with thalidomide but the data are inconclusive (Kennedy et al. 1968, 1975; Vondruska et al. 1971, Shea 1972). Even in the absence of teratogenicity, however, considerable embryotoxicity is often observed (Kennedy et al. 1975). The data on the long-term effects of captan and related fungicides have been critically reviewed by Bridges (1975) who concluded that captan was mutagenic in rats and bacteria, was teratogenic, and had been insufficiently tested for carcinogenesis.

Conclusions:

Captan is eminently suited to soil degradation if well dispersed, rather than massed, in the soil, but since captan is a microbial mutagen no form of unmonitored soil disposal can be recommended. The low persistence of captan in water makes degradation in lagoons a possibility, subject always to the *caveat* of mutagenesis. There are no data available on the feasibility of chemical or thermal destruction of captan, so no conclusions as to the relative merits of different modes of disposal are possible.

Dodine

Dodine is the common name for dodecylguanidine acetate, referred to as doquadine and taitres in France and in the USSR, respectively, and introduced by American Cynamid as Cyprex or Melprex in 1957. Dodine is a white crystalline solid with a melting point of 136°C, soluble in water and ethanol, and insoluble in most organic solvents. The free base is liberated by strong alkalies, but the acetate salt is stable in moderately alkaline as well as moderately acid solutions. Dodine is used chiefly as a foliar fungicide, particularly against apple or pear scab and cherry leaf spot. The usual formulations are 65 percent and 80 percent wettable powders, 75 percent dusts, or 20 percent liquid.

Degradation

The major degradation product of dodine in plants is creatine (Kaufman 1974). Dodine was adsorbed to the same extent by dead as by viable fungal spores; its rapid adsorption and Langmuir-type isotherm suggested ionic bonding to *Alternaria tenuis* spores, while the relative irreversability of its adsorption to *Neurospora crassa* spores suggested covalent bonding. The presence of calcium, magnesium, or uranil salts decreased dodine bonding to fungal spores (Somers and Pring 1966).

Goldberg and Wershaw (1965) found *Achromobacter* and *Flavobacterium* species could grow with dodine as the sole source of carbon. When river mud from the Denver area was treated with dodine, five percent of the fungicide was degraded in 68 days whether or not the mud was preconditioned (Goldberg and Wershaw 1965). There was no information on the chemical, physical or photolytic degradation of dodine.

Transport:

There were no data available on the mobility of dodine in soil, water or air.

Persistence:

There was no data available on the persistence of dodine in soil.

Effects on Non-Target Species:

Dodine was not observed to induce mitotic gene conversion in the yeast *Saccharomyces cerevisiae* under conditions which resulted in gene conversion by both captan and folpet (Siebert et al. 1970).

Shaw (1959) found no difference in honey bee mortality between control and dodine-treated fields. Some reduction in mirid predators was found when dodine was applied to orchards, but 500 ppm dodine in water did not affect adult female parasitic wasps (*Trichogramma*) after a 24-hour exposure (Pimentel 1971).

No data on the effects of dodine on birds, amphibians, fish, or soil fauna were available. The acute oral LD_{50} of dodine was determined to be 566 mg/kg in rats by Jones et al. (1968), and 1,000 mg/kg by Levinskas et al. (1961). The latter calculated a 24 hour dermal LD_{50} of 3,000 mg/kg in rabbits. Chronic feeding of dodine to rats resulted in a reduction in the weight gain of rats fed 3,200 ppm for 100 days or 800 ppm for 2 years, but no effects on reproduction were observed after feeding 800 ppm for 2 years. In dogs, levels of 200 or 800 ppm fed for one year resulted in slight thyroid stimulation. No increase in pituitary chromatophobe adenomas was seen (Levinskas et al. 1961).

Conclusions:

Since no data were available on the persistence of dodine, its nonbiological degradation, or its transport in soil or water, no conclusions can be reliably drawn. The relatively low mammalian toxicity of dodine is reassuring, but its status as a fungicide militates against soil disposal in the absence of information about its toxicity to, and degradation by, soil organisms.

Maneb, Nabam, and Zineb

Maneb is the common name for 1,2-ethanediylbis(carbamodithioato) (2)-manganese, a protective foliar fungicide which was introduced in 1950 by E. I. du Pont de Nemours and Co. as Manzate and by Rohm and Haas as Dithane M-22. Pure maneb is a yellow crystalline solid which decomposes before melting, is slightly soluble in water and insoluble in most organic solvents. Maneb is synthesized by reacting a water-soluble ethylenebis-dithiocarbamate with either manganous sulphate or manganous chloride. The technical product is a light-colored solid. Exposure to either moisture or acids results in decomposition, with formation of polymeric ethylenethiuram monosulfide. Maneb is formulated as a 70 percent wettable powder or in sprays which contain 1.5 to two lbs. per gallon.

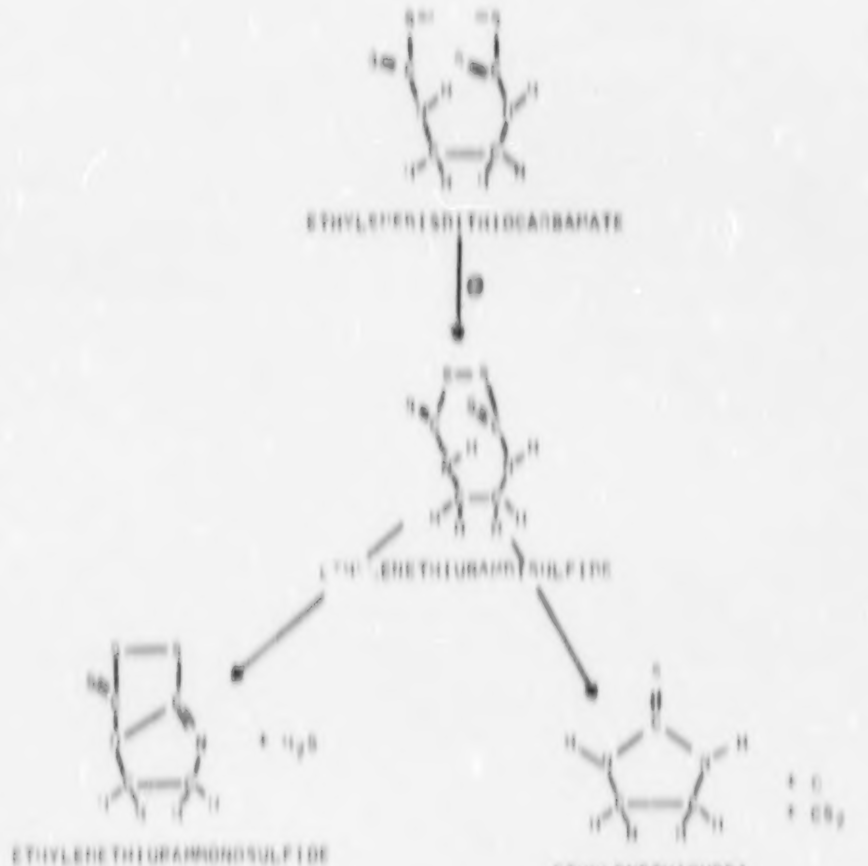
Nabam, the common name for 1,2-ethanediylbis-carbamodithioic acid-disodium salt, was introduced in 1943 by E. I. du Pont de Nemours and Co. as Parzate, and by Rohm and Haas as Dithane D-14. It is synthesized by the interaction of ethylenediamine and carbon disulfide in the presence of sodium hydroxide. It forms colorless crystals of the hexahydrate which are soluble in water to about 20 percent (expressed as the anhydrous salt) at room temperature, forming a yellow solution. Because the crystalline form is unstable, nabam is formulated as a 19 percent aqueous solution. Use of two quarts of this solution to one pound $ZnSO_4$ permits conversion to zineb.

Zineb is the common name for 1,2-ethanediylbis(carbamodithioato) (2)-zinc, was introduced in 1943 as an improvement on nabam and marketed as Dithane Z-78 by Rohm and Haas, and as Parzate-Zineb by E. I. du Pont de Nemours and Co. Zineb is a light-colored powder made by precipitating nabam with soluble zinc salts. It decomposes before melting, has a negligible vapor pressure at room temperature and an aqueous solubility of about 10 ppm at 20°C. It is soluble in pyridine and is somewhat unstable to light, heat, and moisture. The polymer produced by precipitating zineb from concentrated solution has a lower fungicidal activity. Zineb is usually formulated as a 65 percent wettable powder.

Degradation:

Few data on microbial degradation of the dithiocarbamate fungicides exist, in part because of their rapid decomposition in the presence of moisture. *Aspergillus niger* degraded maneb more readily than zineb; almost no water-soluble compounds of the latter were observed (Engst and Schnaak 1967). Figure 18 shows the degradative pathways of the dithiocarbamate fungicides. Essentially identical degradation products are found for all three compounds: ethylenebisthiuram disulfide, ethylenebisthiourea, ethylene diisothiocyanate, CS_2, and H_2S. Munnecke *et al.* (1962) did not find H_2S when nabam was degraded although Klisenko and Vekshtein (1971) reported elemental sulfur. Engst and Schnaak (1970) included ethylenediamine and elemental sulfur among degradation products; the former was reportedly the major fungitoxic product of nabam (Cox *et al.* 1951). In rats, 55 percent of ^{14}C-maneb was excreted as ethylenediamine, ethylene bisthiuram monosulfide and ethylenethiourea (Seidler *et al.* 1970).

In the laboratory, zineb lost its efficacy in 16 days at 30° or 40°C. In the field, the fungitoxicity of zineb increased for four days, then declined with 50 percent of its bioactivity gone after 16 days (Grandi *et al.* 1958). It is noteworthy that the degradative products of the dithiocarbamates are probably the active fungicides. Rich and Horsfall (1950) observed that SO_2, H_2S, and ethylene thiourea accounted for only part of the fungitoxicity of nabam; Engst and Schnaak (1967) considered ethylenethiuram monosulfide to be the chief fungitoxin of the dithiocarbamate fungicides. Engst (1970) noted the greater toxicity of some metabolites of these fungicides, and cited ethylenebis(thiouronium sul-


ETHYLENEBISDITHIOCARBAMATE
ETHYLENETHIURAMDISULFIDE
+ H_2S
ETHYLENETHIURAMMONOSULFIDE
+ S
+ CS_2
ETHYLENETHIOUREA

DEGRADATION OF ETHYLENEBISDITHIOCARBAMATES
(AFTER HYLIN 1973)

Figure 18

fide) as the major fungitoxin. Viel and Chancogne (1966) tentatively identified the fungicidally active product of maneb as ethylenethiuram monosulfide.

Zineb was more rapidly degraded in soil than in liquid cultures. Exposure to sunlight for 19 months did not affect its degradation (Iley and Fiskell 1963). Zineb was decomposed by temperatures of 200°C, with a weight loss of 34 percent and a change in color to brownish-black (Stojanovic et al. 1972b). At 900°C, the volatile products of zineb decomposition were CO, CO_2, H_2S, and NH_3 (Kennedy et al. 1972b).

Chemically zineb could be degraded 99 percent or more by treatment with liquid NH_3 plus metallic sodium or lithium, or with sodium biphenyl, while triethanolamine was not effective. Treatment with 18N H_2SO_4 resulted in formation of H_2S and free zinc (Kennedy et al. 1972a). Nabam in soil was inactivated nonbiologically and very rapidly (Munnecke 1958).

Transport:

In soil columns, mobility was more affected by soil type for fungicides in suspension, such as zineb, than for those in solution, such as nabam. Dissolved fungicides were more mobile than suspended fungicides, and mobility of all fungicides was enhanced in wet rather than dry soil and in sandier and/or more porous soil. Peat moss greatly decreased mobility (Munnecke 1961). No data were available on the transport of maneb, nabam, or zineb into or within water. Ethylenethiourea was taken up by cucumbers (Vonk 1971).

Persistence:

As suggested by the ready decomposition of the dithiocarbamates by moisture, persistence in soil appears to be low. Maneb persisted for three weeks when applied at one ppm, and more than 11 weeks but less than 12 weeks when applied at 1,000 ppm (Chinn 1973). No data were available on the persistence of the intermediate degradation products of the dithiocarbamate fungicides in soil. When beans and tomatos were treated at seven day intervals with maneb with seven and eight treatments respectively, residues were present on the plants 14 days later (Newsome et al. 1975).

Effects on Soil and Soil Organisms:

The effects of maneb, nabam, and zineb on soil processes and soil microorganisms are summarized in Tables 87, 88 and 89. It can be seen that, despite their economic status as fungicides, the dithiocarbamates are not uniformly or universally toxic to soil organisms. Even relatively rapid recovery of fungal numbers (within 60 days) has been reported (Chandra and Bollen 1961) but after nabam treatment, soil fungal populations consisted mostly of *Trichoderma* and *Penicillium* (Gordon and Young 1965).

Dubey and Rodrigues (1970) analysed the effects of nabam on nitrification and ammonification and found that nitrification was less inhibited in rapidly nitrifying loam soil than in slowly nitrifying lateritic soil. The ammonia-oxidizing bacteria were most strongly affected, while nitrite-oxidizing bacteria were not inhibited. Maneb at 15 or 60 ppm inhibited nitrification for eight weeks, while ammonification was inhibited only at 960 ppm. Zineb applied to calcareous loam (pH 7.5) at 5,000 ppm reduced CO_2 evolution by 86 percent over a 56 day observation period (Stojanovic et al. 1972a). Mutagenesis was reported in *Alternaria mali* by maneb, nabrm and zineb (Slifkin 1973), and in *Salmonella* by maneb (Seiler 1973) but maneb was reportedly not mutagenic in *Saccharomyces* (Siebert et al. 1970).

Effects on Non-Target Species:

No evidence of mutagenesis by zineb was found in two studies on *Drosophila melanogaster* (Pilinskaya 1967, Ryazanov 1967). Zineb was essentially nontoxic to honeybees at even the highest agricultural levels of exposure (Nazarov 1966).

Table 87. THE EFFECTS OF MANEB ON SOIL ORGANISMS OTHER THAN FUNGI

Parameter	Effect	Level Original	Level PPM	# days Observed	Soil or conditions	Reference
Ammonification	-		960	56	loam; lateritic	Dubey and Rodriguez 1970
Nitrification	-		15	56	loam; lateritic	Ibid.
Ammonification	+	0.1%	1000			Damer and Zarova-Tomaleva 1968
Soil microorganisms	-	0.1-0.5%	1000-5000			Ibid.
Denitrifying bacteria	0	0.1-0.5%	1000-5000			Ibid.
Bacteria	+	60 ppm	60	<300	alkaline loam	Dubey and Rodriguez 1974
	+	240 ppm	240	<300	alkaline loam	Ibid.
	+	960 ppm	960	540	loam or clay	Ibid.
	+	>240 ppm	>240	300	acid clay	Ibid.
Actinomycetes	-	960 ppm	960	14	clay or loam	Ibid.
Euglena gracilis	-			2 hours	culture	Moore 1970

+ = stimulation
0 = no effect
- = inhibition

Table 88. THE EFFECTS OF NABAM ON SOIL ORGANISMS OTHER THAN FUNGI

Parameter	Effect	Level		# days		
		Original	PPM	Observed	Soil or conditions	Reference
Phytoplankton	–	10 ppm	10		seawater	Ukeles 1962
Dunaliella	–		1		seawater	Ibid.
Phaeodactylum	–		1		seawater	Ibid.
Monochrysis	–		1		seawater	Ibid.
Bacteria	0					Domsch 1959
Actinomycetes	0					Ibid.
Algae	0					Ibid.
Bacteria					bean rhizosphere	Hallock and Cochrane 1950
Bacteria	–	38 lbs/A	19	180	4 Oregon soils	Chandra and Bollen 1961
Nitrification	–	38 lbs/A	19	60	4 Oregon soils	Ibid.
CO_2 evolution	–	38 lbs/A	19	28	4 Oregon soils	Ibid.
Euglena gracilis	–	10 ppm	10	2 hours	culture	Moore 1970

+ = stimulation
0 = no effect
– = inhibition

Table 89. THE EFFECT OF ZINEB ON SOIL ORGANISMS OTHER THAN FUNGI

Parameter	Effect	Level		# days		
		Original	PPM	Observed	Soil or conditions	Reference
Bacteria	0	≤500 ppm	≤500		soil	Hubbell *et al.* 1973
Actinomycetes	0	≤500 ppm	≤500		soil	Ibid.
Nitrification	0	≤500 ppm	≤500		soil	Ibid.
Nitrification	-	≥500 ppm	≥500		silt loam	Wilson 1954
CO_2 evolution	-	5 tons/A	5000	56	calcareous loam	Stojanovic *et al.* 1972a
Euglena gracilis	+	1.25	1.25	1 hour	culture	Moore 1970

+ = stimulation
0 = no effect
- = inhibition

In the amphibian *Xenopus laevis*, nabam and maneb affected growth, melanogenesis, and notochordal development when eggs were exposed to 1-5 ppm for 36-38 hours before hatching (Bancroft and Prahlad 1973, Prahlad *et al.* 1974). In hens, treatment with one percent of the LD_{50} of zineb decreased fertility, impaired egg development and caused degenerative changes in hens' livers, kidneys, and reproductive organs (Zhavoronkov *et al.* 1973).

The oral LD_{50} of dithiocarbamate fungicides to rats was calculated to be between 1,000 and 8,000 mg/kg (Jones *et al.* 1968). Inhalation of zineb was observed to cause liver and kidney damage in albino rats (Ivanaova 1966). A single dose of not more than 500 mg/kg or ten doses of 100 to 250 mg/kg of zineb did not cause poisoning in sheep (Palmer and Radeleef 1964). Nabam pretreatment decreased the acute toxicity of methyl parathion in mice (Lange *et al.* 1975). Maneb and zineb, if given at 250 mg/kg/day, reportedly increased blood coagulability in rabbits (Karpenko 1975).

In cell culture, the carbamates zineb and carbaryl were found to accumulate more readily than the organochlorines, HCH and DDT with lethal levels markedly lower in culture than *in vivo* (Shpirt 1973). Zineb was considered to affect cell mitotic activity and cause chromosome damage (Rashev 1972); similar results were obtained when mice were injected with maneb or zineb (Kurinnyi and Kondratenko 1972).

Ethylene thiourea is a known thyroid carcinogen (Bontoyn and Looker 1973, Hylin 1973) and it also caused lung tumors in rats (Rappoport *et al.* 1966) or orally (Didenko and Dupalovich 1975, Chernov and Khitsenko 1969, Chernov 1972, Andrianova adn Alekseev 1970, Engst and Schnaak 1974). Maneb caused lung adenomas in mice (Chernov 1972) and in rats (Balin 1970). Chepinoga *et al.* (1970) administered 0.17 times the LD_{50} of zineb to rats, and classified the compound as blastemogenic and embryotoxic while in the same study, maneb was found to be strongly embryotoxic and weakly mutagenic. Zineb was not mutagenic and maneb was not tested for carcinogenicity. Zineb was a transplacental carcinogen in mice (Kuitnitskaya and Kolesnichenko 1971).

Ethylene thiourea, a degradation product of the thiocarbamate fungicides, was teratogenic and embryocidal in rats but only embryocidal in rabbits (Khera 1973). Maneb was embryocidal in rats, and also caused reversible sterility of both males and females (Martson 1969). Maneb and zineb were teratogenic in rats only at levels greater than 2 g/kg, leading Petrova-Vergieva and Ivanova-Chemishanska (1971, 1973) to conclude that human teratogenesis was unlikely.

Conclusions:

All methods of disposing of the dithiocarbamate fungicides maneb, nabam and zineb must take into account the carcinogenic metabolite ethylene thiourea. If and only if this compound can be shown to be non-persistent in soil is soil disposal of these compounds feasible. Either chemical or thermal decomposition requires caution because of the release of H_2S, NH_3, and/or CO, but both are feasible for the dithiocarbamate fungicides.

Methyl Bromide

Methyl bromide is the common name for bromomethane, a colorless gas with a boiling point of 4.5°C and a freezing point of -93°C. The colorless liquid has an odor similar to that of chloroform. Its solubility in water at 25°C is 13,400 ppm and it forms a voluminous crystalline hydrate with ice water. It is stable, neither corrosive nor inflammable, and soluble in most organic solvents. Methyl bromide is synthesized by the action of hydrobromic acid on methanol. In the United States it is produced by American Potash, Dow Chemical Co., Frontier Chemical Co., Great Lakes Chemical Co. and Michigan Chemical Co. for use as a soil fumigant against nematodes, fungi, and weeds. It is also used in the fumigation of storage areas and of stored products.

Degradation:

Methyl bromide is decomposed by oxidative processes in soil and/or conjugated with sulfhydryl-containing compounds, with concomitant debromination (Goring et al. 1975). Shiroishi and co-workers (1964) used ^{14}C-methyl bromide to fumigate soil and found the residual radioactivity after one month to be heavily associated (44 percent) with protein. The same study demonstrated conjugation of methyl bromide with free amino acids.

In the absence of oxygen, methyl bromide heated to 550°C decomposed to form methane (20 percent), hydrogen bromide (45 percent), considerable quantities of hydrogen, bromine, bromoethane, and CH_2. Other products included $(CH_2Br)_2$, anthracene and pyrene (Chaigneau et al. 1966).

Photolysis of methyl bromide produced CH_4 when carried out in the presence of silver, copper, or gold at temperatures between 50° and 250°C. Some C_2H_6 was also recovered (McTigue and Buchanan 1959). Photolysis of methyl bromide at 1,850 A at 25 to 30°C in the presence of three percent bromine resulted in formation of methane (Kobrinsky and Martin 1968).

Transport-

Methyl bromide is primarily lost into the air after soil fumigation. Some adsorbed methyl bromide might move with or through soil, but no data were available. Soil fumigation with 73 g/m^3 of methyl bromide resulted in residues of up to 45 ppm in mature tomatoes, although the usual residues were considerable lower (Kempton and Maw 1973). Stored beans retained very high levels of methyl bromide for more than 17 days after treatment (Seefeld and Beitz 1968). Aged methyl bromide was less readily taken up by plants than fresh, and plants accumulated more methyl bromide in roots and fruit than in leaves or stalks (Malkomes 1972).

Persistence-

Methyl bromide is considered a nonpersistent pesticide inasmuch as more than 50 percent disappears from soil within one-half month (Goring *et al*. 1975). Sorption of methyl bromide was greatest in peat, least in sand, and intermediate in clay soils; all soils adsorbed more methyl bromide when dry. Moisture did not, however, increase the loss of methyl bromide from empty chambers (Chisholm and Koblitsky 1943). In sandy clay soil, eleven percent moisture affected little reduction of methyl bromide in a one-hour period (Fuhr *et al*. 1968). The depth of soil penetration by methyl bromide depended on the dose applied (Malkomes 1972). Food residues of methyl bromide decreased with decreasing temperatures during fumigation (Dumas 1973)

Effects on Non-Target Species-

The toxicity of methyl bromide to soil organisms can be gauged by its use as an herbicide, fungicide and nematocide (Martin 1968). Essentially complete extermination of soil fauna in pine litter followed treatment with 25 g/m^2 (250.4 kg/ha) of methyl bromide (Heungens 1972). Among bacteria, spore forming organisms were more resistant than nitrifying bacteria and fungi were more sensitive than actinomycetes. Methyl bromide was both less toxic and less selective in its toxicity than chloropicrin (Reber 1967). Toxicity was greater in wet than in dry soils (McClellan et al. 1974).

In fine sand or fine loamy sand, 2 lbs/100 ft^2 (978 kg/ha) of methyl bromide decreased nitrification for 50 days, but overall bacterial activity increased above pretreatment levels after three weeks, and numbers of *Trichoderma* exceeded pretreatment levels after 30 weeks (Overman 1972). Winfree and Cox (1958) observed a two-month inhibition of soil nitrification in Everglades peat after treatment with methyl bromide at 2 lbs/100 ft^2 (978 kg/ha).

Jenkinson and Powlson (1970) were able to detect effects of the elimination of a section of the soil biomass by methyl bromide years later by stressing the soil organisms with formalin or radiation. Second and subsequent fumigation resulted in less nitrogen mineralization regardless of the populations of pathogens present. These data suggest permanent effects of methyl bromide on the complex of soil microorganisms even after the ordinary parameters of soil effects have returned to normal.

In the host-mediated assay in mice, methyl bromide was mutagenic for *Salmonella typhimurium* and *Salmonella marcescens* (Buselmaier et al. 1972).

Conclusions:

As a gas, methyl bromide would be totally unsuitable for soil disposal even if its toxicity to soil microorganisms were less profound or less long-lasting. A reasonable method of disposal is suggested by the ready anoxic decomposition of methyl bromide at 550°C.

Pentachlorophenol

Pentachlorophenol is a white crystalline solid which is only slightly soluble in water (20 ppm at 30°C) but soluble in most organic solvents. It has a melting point of 190°C and a boiling point of 293°C. Its vapor pressure increases from 0.00011 mm Hg at 20°C to 0.12 mm Hg at 100°C, and it is volatile in steam. Technical pentachlorophenol is a dark grey powder with a melting point between 187 and 189°C.

Pentachlorophenol was introduced as a wood preservative in 1936 and is used as a fungicide, herbicide, defoliant and insecticide. The sodium salt of pentachlorophenol is readily water soluble (300,000 ppm water at 25°C), but insoluble in petroleum oils. It is used as a molluscicide and in other situations requiring aqueous solutions. Pentachlorophenyl laurate, a nonfungicidal analog of pentachlorophenol, has been used as a mothproofing compound (Adema et al. 1967).

Pentachlorophenol, produced by the catalytic chlorination of phenol, is manufactured by Dow Chemical Co. as Dowicide 7 and by Monsanto Chemical Co. as Santophen 20. The corresponding sodium pentachlorophenate products are Dowicide G and Santobrite. The technical product is used directly or is formulated in oil. Although pentachlorophenol is not deemed to need a trivial name, it is frequently referred to as PCP.

Degradation:

Biological--Pentachlorophenyl laurate was hydrolyzed to pentachlorophenol in the presence of soil, presumably by microbial action (Allsopp et al. 1970). Degradation of pentachlorophenol by *Trichoderma virgatum* was observed, but no metabolites were identified (Cserjesi 1967a). A single strain of *Aphaloascus fragrans* was acclimatized to 0.2 percent (2,000 ppm) pentachlorophenol; all other species tested (*Tricho-*

derma harzianum, Trichoderma virgatum, Trichoderma viridi, Ceratocystis pilifera, Chaetomium globosum, Graphium sp., *Penicillium* sp.) were inhibited by 0.04 percent (400 ppm) pentachlorophenol (Cserjesi 1967b).

Lyr (1963) reported detoxification of chlorinated phenols by fungal oxidases, but no ring cleavage occurred, and the more highly chlorinated compounds were less susceptible even to detoxification. In paddy soil, pentachlorophenol decomposed to mono-, di-, tri-, and tetrachlorophenol. Decomposition was more rapid in mature paddy soil than in immature soil with higher levels of volcanic ash. Since soil sterilization inhibited decomposition, microbial action was presumed (Ide et al. 1972).

Suzuki and Nose (1970) analyzed decomposition of pentachlorophenol in farm soils, and observed that 90 percent was detoxified in ten days when 100 ppm were applied to the soil, but only ten percent detoxification of 1,000 ppm occurred in the same period. Chloropicrin (Cl_3CNO_2) and C_6H_6HgO-acetate enhanced detoxification, whereas captan was inhibitory. A gram-positive bacillus converted PCP to pentachloroanisole, with hydroquinone dimethyl ether as a minor product (Suzuki and Nose 1971). Chu and Kirsch (1972) reported that a species of saprophytic coryneform bacteria was able to grow using pentachlorophenol as the sole source of carbon. No other reports of ring cleavage were found. Vel-Muzquiz and Kaspar (1974) considered microbial degradation to be relatively insignificant in the detoxification of pentachlorophenol, since they could not find evidence that microorganisms could grow with PCP as the sole source of carbon.

Chicken-house litter treated with pentachlorophenol imparted a musty taint to the chicken meat due to the microbial conversion of PCP to 2,3,4,6-tetrachloroanisole (Curtis et al. 1972). In rats and mice, tetrachlorohydroquinone was the major, if not only, metabolite (Jakobsen and Yllner 1971, Ahlborg and Lindgren 1974).

Photolytic--Sodium pentachlorophenate was inactivated by light of less than 330 nm when tested by bioassay against snail eggs (Hiatt *et al.* 1960). Pentachlorophenol itself was, in contrast, very stable to light (Crosby and Hamadmad 1971). Among numerous compounds, including oxidized monomers and dimers, produced by the action of light on sodium pentachlorophenate were five products with greater toxicity to fish. Chloranilic acid was also produced, lending a purple color to the solution (Munakata and Kuwahara 1969). Under field conditions, sodium pentachlorophenate was more rapidly degraded in clear, shallow water than in deep, turbid water, (Bevenue and Beckman 1969) suggesting that photolysis is a major degradative route.

When sodium pentachlorophenate was exposed to sunlight, traces of octachlorodibenzo-*p*-dioxin and other unidentified dioxins were found. The [illegible] were unstable, but octachlorodibenzo-*p*-dioxin was stable to ultraviolet (Stehl *et al.* 1973, Plimmer 1973).

In the presence of a large excess of oxygen, pentachlorophenol exposed to UV light of 230 nm or 290 nm was decomposed with 69 mg of the initial 80 mg remaining after seven days. In addition, 15 mg CO_2 and six mg HCl were also recovered (Gaeb *et al.* 1975).

Chemical and physical--Pyrolysis of PCP at $300^{\circ}C$ for 24 hours resulted in conversion to hexachlorobenzene, octachlorodiphenylene dioxide and other neutral polymeric products (Sanderman *et al.* 1957). Slow pyrolysis at $200^{\circ}C$ resulted in production of octachlorodibenzodioxin, while conversion of sodium pentachlorophenate to dioxins occurred at about $360^{\circ}C$ (Langer *et al.* 1973). Nitric acid converts pentachlorophenol to a mixture of tetrachloro-*o*-and *p*-quinones (Bevenue and Beckman 1967).

Transport--

Pentachlorophenol was relatively immobile in acidic Hawaiian soil, but increasingly mobile as pH increased (Green and Young 1971). In sandy paddy soil, sodium pentachlorophenate leached more readily than PCP (Asa and Sakamoto 1963). PCP soil penetration was greater in xylenol emulsions than in oil emulsions (Sakagami *et al.* 1963).

Transport of pentachlorophenol or its salts into water was demonstrated by the presence of 0.1 to 0.7 ppb of pentachlorophenol in the Willamette River above Corvallis while the effluent from sewage treatment plants in three Oregon cities contained one to four parts per billion PCP in the water (Buhler *et al.* 1973).

Persistence-

The effectiveness of PCP decreased with decreasing pH between pH 3 and pH 8 and also decreased with increasing organic matter content of the soil and with increasing soil surface area (Su and Lin 1970). Adsorption to soil was positively correlated with soil organic matter, pH, and cation exchange capacity, and negatively correlated with clay content and bioactivity (Tsunoda 1965). Choi and Aomine (1972) also noted that pentachlorophenol was less effective and more persistent in acid than in neutral soil, but there was no correlation in their study between activity and the amount of clay, type of clay, or cation exchange capacity of the soil once the effects of pH and solubility were eliminated. Low pH apparently decreased toxicity by decreasing the solubility of pentachlorophenol. Sodium pentachlorophenate was less soluble in humus-rich than in mineral soils, as would be expected of a lipid-immiscible compound.

Treatment with 15 kg/ha of technical pentachlorophenol resulted in soil residues of 20.6 ppm of pentachlorophenol and 0.5 ppm of its impurity, hexachlorobenzene. The corresponding residues in lettuce were 0.73 ppm and 0.01 ppm of PCP and hexachlorobenzene, respectively (Casanova and Dubroca 1973). No degradation of either pentachlorophenol or sodium pentachlorophenate was observed after twelve months when these compounds were applied to warm, moist soil, but fixation to the clay fraction of the soil was not found (Harvey and Crafts 1952). Pentachlorophenol applied to soil as a termiticide at an unspecified level persisted for more than five years (Hetrick 1952).

Effects on Non-Target Species—

Microorganisms—Data on the toxicity of pentachlorophenol to microorganisms are summarized in Table 90. On soil derived from volcanic ash, pentachlorophenol was the strongest inhibitor of nitrification among eight herbicides tested. In increasing order of toxicity, the compounds were: Prometryne < lenacil < diphenamid < trifluralin < vernolate < MCPA < SWEP < pentachlorophenol. Pentachlorophenol was the only compound to prevent oxidation of nitrite to nitrate as well as nitrite formation from ammonium ion (Noguchi and Nakazawa 1971).

Invertebrates—Pentachlorophenol applied at 2.5 to 4 kg/ha decreased the number of earthworms (species not identified) in the top ten centimeters of soil by 50 percent (Kononova and Kazimirchuk 1970). In sea urchin eggs (*Arbacia*), pentachlorophenol inhibited mitoses and disturbed the structure of the mitotic spindle (Sawada and Rebhun 1969). No embryos of clams (*Mercenaria mercenaria*) or oysters (*Crassostrea virginica*) exposed to 0.25 ppm sodium pentachlorophenate survived, nor did eggs of either species hatch when exposed to 0.025 ppm sodium pentachlorophenate. At the lower dose, 41 percent of oyster larvae survived (Davis and Hidu 1969).

Vertebrates—The 24 hour LC_{50} of sodium pentachlorophenate for rainbow trout (*Salmo gairdneri*) was 0.26 ppm; for guchi fish (*Nibea albiflora*), 0.09 ppm (Pimentel 1971). Bevenue and Beckman (1967) reviewed the toxicology of pentachlorophenol to fish.

In birds, the LD_{50} for five days' treated feed followed by three days' clean feed was 4,000 to 5,000 mg/kg pentachlorophenol in pheasants, and 5,000 to 6,000 mg/kg in coturnix quail (Pimentel 1971).

The acute oral LD_{50} of pentachlorophenol was 27 to 80 mg/kg in rats, while that of sodium pentachlorophenate was 210 mg/kg (Pimentel 1971).

Table 90. EFFECTS OF PENTACHLOROPHENOL ON SOIL MICROORGANISMS

Parameter	Effect	Dose		Soil/conditions	Reference
		Original	PPM		
CO_2 evolution	–	2 lbs/A		soil in flasks	Kratochvil 1951
	–	≤5000 ppm	≤5000	clay-loam + manure	Matsuguchi 1962
Nitrification	–	200 ppm	200	nitrite from ammonium	Noguchi and Nakazawa 1951
	–	200 ppm	200	nitrate from nitrite	Ibid.
Bacteria, all	–			cultures	Langkramer 1970
	–			field	Vel-Munquiz and Kamper 1974
Sporulating	–			cultures	Langkramer 1972
Aerobic cellulolytic	0	333 l/ha			Pochon et al. 1951
Nitrifying	+	333 l/ha			Ibid.
Azotobacter	–	333 l/ha			Ibid.
	–				Langkramer 1970
Bacilli	–				Ibid.
Fungi	–				Ibid.
	–				Vel-Munquiz and Kamper 1974
Actinomycetes	–				Ibid.

+ = stimulation
0 = no effect
– = inhibition

Tilemans and Dormal (1952) cited a human acute oral LD_{50} of 275 mg/kg. Iseke and Iwao (1956) calculated an acute oral LD_{50} of 199 mg/kg for mice. Dermal toxicity is also well known (Bevenue and Beckman 1967).

Pentachlorophenol was markedly less toxic in rabbits at 8°C than at 36°C (Kaplinger *et al.* 1959). The biochemical basis of the toxicity of pentachlorophenol is its capacity to uncouple oxidative phosphorylation at concentrations 10^{-6} to 10^{-4}M. Between 10^{-4}M and 10^{-3}M, pentachlorophenol also inhibits mitochondrial ATPase, and at levels above 10^{-3}M, PCP inhibits glycolytic phosphorylation, inactivates respiratory enzymes, and causes gross damage to mitochondrial structures (Weinbach 1957). No data pertaining to carcinogenesis, mutagenesis, or teratogenesis of PCP were available.

Conclusions:

Sufficiently little is known about the degradation of pentachlorophenol that any method of disposal is a gamble. The few available data make it obvious, however, that biodegradation rarely includes ring cleavage. Soil disposal is therefore inadvisable unless the nature, toxicity, and persistence of the terminal residues are elucidated. Similarly, in the absence of extensive data on the nature, quantity, toxicity, and persistence of the dioxins produced by pyrolysis of PCP over a wide range of temperatures, no conclusions can be drawn as to the feasibility of burning this compound.

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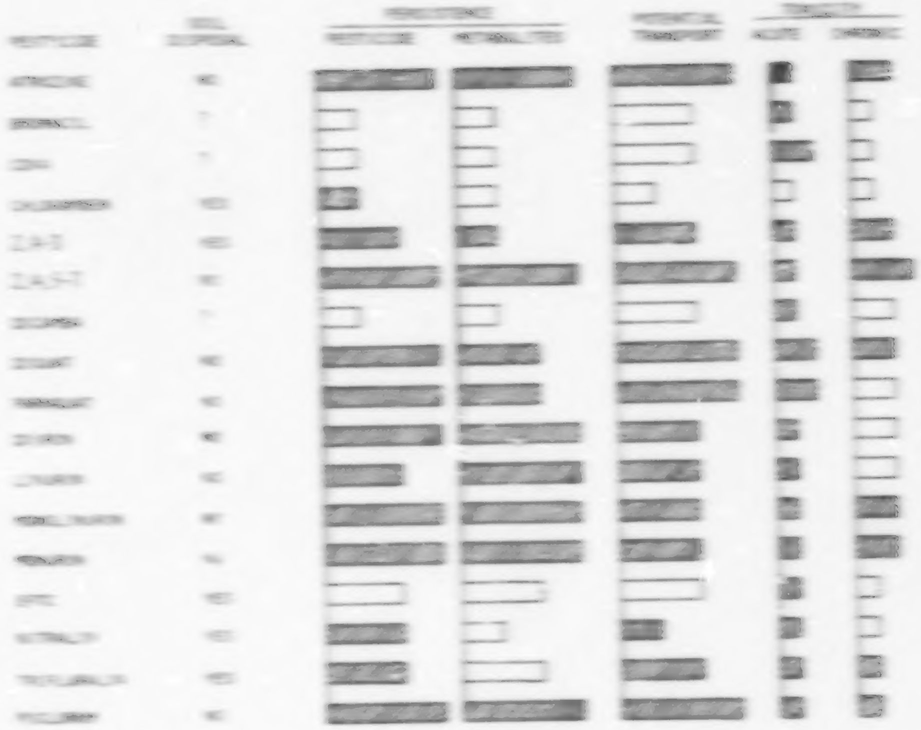
SECTION VI SYNTHESIS

Figures 19, 20 and 21 summarize the data available for the 45 pesticides reviewed in this report. Persistence of both the original pesticide and of its metabolites or contaminants were characterized as low, moderate or high by the length of the bar on each graph, and the potential transport was similarly ranked. These were considered the primary criteria for each pesticide's suitability for soil disposal. Evaluation of toxicity is included in the figures for completeness, as is the conclusion as to the feasibility of soil disposal, which is repeated from Table 1.

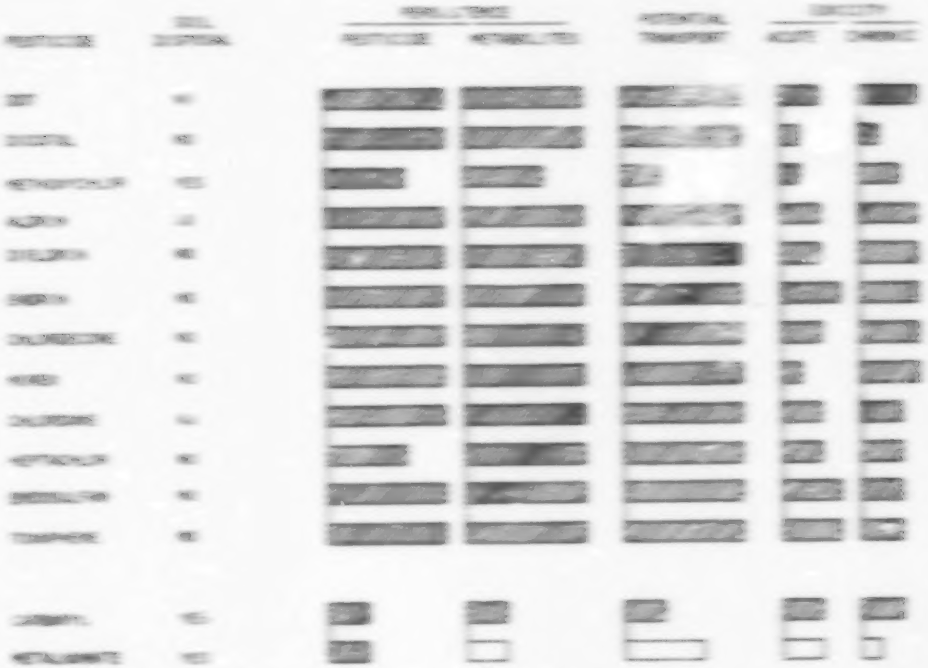
The acute toxicity of a pesticide includes its effects on soil organisms, aquatic organisms, birds and mammals, while chronic toxicity includes reproductive effects and carcinogenesis. The characterization of persistence, transport and toxicity is qualitative by necessity. Where the available information is too incomplete even for qualitative conclusions, unshaded bars were used.

Of forty-five pesticides, only ten are considered suitable for soil disposal in the light of presently available data. For several of these, suitability is presumed despite very little information because of the absence of unfavorable information. Several organophosphate compounds (methyl parathion, parathion, azinphosmethyl) are considered questionably soil disposable because recent data suggest they do not decompose when massed in soil. Therefore it must be stressed that the characterization of pesticides as suitable for soil disposal is optimistic, and the number of such compounds will probably decrease as more data accu-



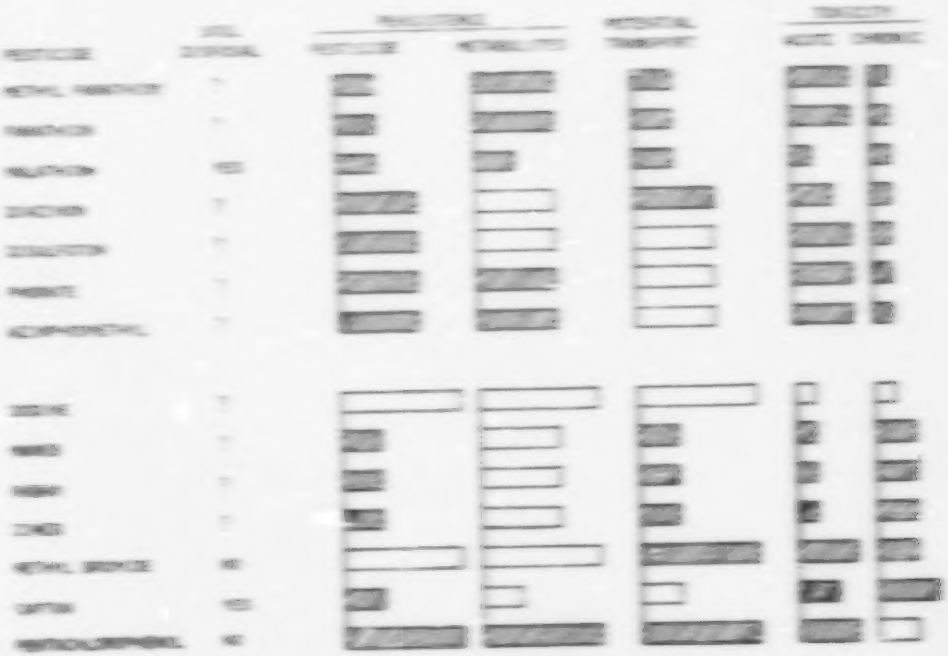
FEASIBILITY OF SOIL DISPOSAL OF HERBICIDES: EVALUATION OF AVAILABLE DATA

Figure 19



FEASIBILITY OF SOIL DISPOSAL OF CHLORINATED HYDROCARBON AND CARBAMATE INSECTICIDES: EVALUATION OF AVAILABLE DATA

Figure 23



FEASIBILITY OF SOIL DISPOSAL OF ORGANOPHOSPHATE INSECTICIDES AND MISCELLANEOUS PESTICIDES - EVALUATION OF AVAILABLE DATA

Figure 21

mulate. In contrast, there is a small but carefully established body of data on chemical and thermal disposal of certain pesticides. Although incineration of pesticides requires many precautions, it seems more effective, and certainly more complete, than soil disposal.

We therefore conclude that incineration is a more promising mode of pesticide disposal than is soil incorporation, and strongly recommend its continued assessment.

APPENDIX

The data cited in this report are cited in metric units both in the tables and in the text. Where the data were originally reported in other than metric units, the following conversions were employed:

soil treatments:	1 lb/A = 1.12 kg/ha
soil concentrations:	0.5 ppm = 1.0 kg/ha
solutions:	1.0% = 10,000 ppm
	1 mg/ml = 1,000 ppm